

A 93-Year-Old Woman with an Abnormal Electrocardiogram

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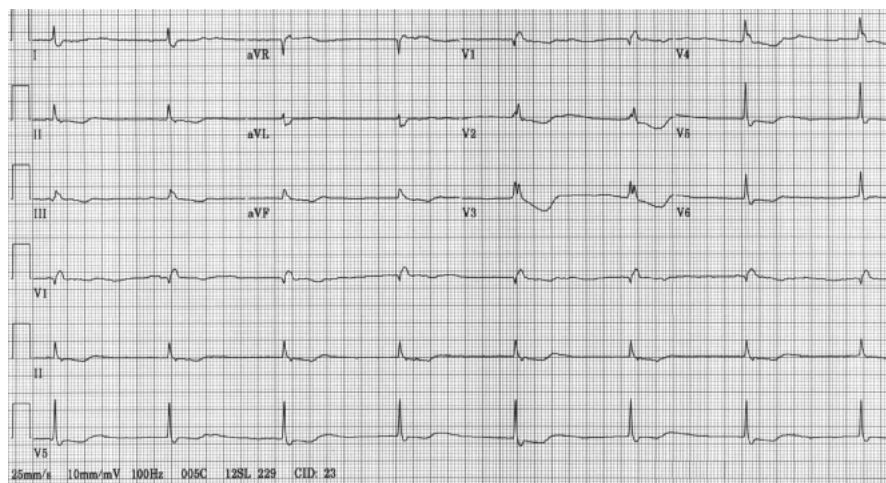


Figure. Electrocardiogram of the case patient on presentation.

CASE PRESENTATION

A 93-year-old woman presented to the outpatient clinic with a chief complaint of fatigue for the past week. She denied chest pain, shortness of breath, nausea, vomiting, diarrhea, abdominal pain, headache, or any visual disturbances. Her past medical history included hypothyroidism, hypertension, atrial fibrillation, and breast cancer, for which she had received radiation therapy. She had been recently diagnosed with sternal skin cellulitis and was being treated with ciprofloxacin. Two years prior to the current presentation, an electrocardiogram (ECG) showed a complete right bundle branch block (RBBB) with normal sinus rhythm. Two days prior to her admission, a computed tomography scan of the chest with contrast was performed to evaluate the extension of the cellulitis and the presence of any breast metastasis. Her current medications were levothyroxine, tamoxifen, and digoxin.

On physical examination, the patient had a heart rate of 45 bpm with a regular rhythm. Her blood pressure was 106/65 mm Hg. S_1 was normal, and S_2 was

split. There were no murmurs or gallop. Jugular venous distention was not present. Her chest examination was normal, and the sternal skin cellulitis was healing well. Initial laboratory results are listed in **Table 1**. The patient had a baseline creatinine level of 1.2 mg/dL, and her most recent echocardiogram showed normal systolic function and an ejection fraction of 60%. An ECG was obtained, and is shown in the **Figure**.

WHAT DOES THE ECG DEMONSTRATE?

- (A) Normal sinus rhythm with complete RBBB
- (B) Junctional rhythm with complete RBBB
- (C) Left ventricular hypertrophy (LVH)
- (D) Accelerated junctional rhythm with RBBB

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Table 1. Laboratory Values of the Case Patient on Presentation

Variable	Result	Reference Range
Sodium (mEq/L)	133	135–146
Potassium (mEq/L)	5.0	3.5–5.5
Chloride (mEq/L)	105	95–112
Carbon dioxide (mEq/L)	23	22–32
Urea nitrogen (mg/dL)	70	7–25
Creatinine (mg/dL)	4.0	0.7–1.4

WHAT IS THE MOST LIKELY CAUSE?

- (A) Myocardial ischemia
- (B) Hyperkalemia
- (C) Hypertension
- (D) Digoxin toxicity

ANSWERS

The correct answers are (B) junctional rhythm with complete RBBB, and (D) digoxin toxicity.

DISCUSSION

The ECG demonstrates junctional rhythm with RBBB. Junctional rhythms are escape rhythms that can occur when the sinoatrial (SA) node is no longer functioning, when the patient has marked bradycardia, or when there is a high-degree atrioventricular (AV) block. A normal junctional rhythm is defined as a rate between 40 and 60 bpm. When the junctional rhythm is above 60 bpm, it is called an accelerated junctional rhythm.¹

LVH is diagnosed if one or more of the following criteria are met¹:

- In the limb leads: R wave in lead I + S wave in lead III > 25 mm (11% sensitive), or R wave in aVL > 11 mm (11% sensitive)
- In the precordial leads: R wave in V₅ or V₆ > 26 mm (25% sensitive), or R wave in V₅ or V₆ + S wave in V₁ > 35 mm (43% sensitive)

The case patient's ECG findings do not meet the criteria for LVH.

Digoxin toxicity can present with a wide variety of ECG abnormalities. The suspicion is heightened when there is evidence of enhanced automaticity and decreased conduction, as in the case patient. The sudden change from the irregular rhythm of atrial fibrillation to a slow, regular rhythm (a junctional rhythm) is suggestive of digoxin toxicity. The case patient was taking digoxin as directed, and her digoxin level at admission was 2.7 ng/mL (reference range, 0.7–2.0 ng/mL).

The presence of RBBB in this patient complicates the interpretation of ST elevation in leads V₁ through V₃. The diagnosis of acute anterior myocardial infarction (MI) is still possible when the patient has secondary T wave changes in leads V₁ through V₃; that is, when the usually discordant T waves (opposite to the QRS complex) become concordant in these leads.¹ The presence of RBBB does not interfere with the interpretation of ST elevation in other leads, nor does it interfere with the recognition of Q waves in the setting of an MI. The previous ECG of this patient shows she has a pre-existing RBBB, making myocardial ischemia less likely; in addition, she does not now have any Q waves or any changes in the ST segments or T waves that are suggestive of myocardial ischemia or infarction and her cardiac enzymes were also normal.

Hyperkalemia can present on the ECG as tall, peaked T waves. Hyperkalemia can also cause interventricular conduction defects. The QRS becomes prolonged, especially if the serum potassium level is greater than 6.5 mEq/L. Widening of the QRS complex is diffuse, and, as the widening progresses, the QRS complex may appear as “sine waves.” Hyperkalemia accelerates repolarization and lowers the resting membrane potential; this depresses AV nodal conduction and can lead to complete heart block in the presence of digoxin toxicity. This typically occurs with potassium levels greater than 6.0 mEq/L. The case patient had a potassium level of 5.0 mEq/L, which is within the normal range; in addition, she had no ECG changes suggestive of hyperkalemia.²

The ECG is normal in most patients with hypertension; however, abnormal ECG findings that may accompany hypertension are LVH and left atrial enlargement, with or without abnormalities in the conduction system, including RBBB.¹ Fibrosis has been found in pathology studies of patients with RBBB,³ and it is known that hypertension is associated with myocardial fibrosis.⁴ This may be especially true in patients younger than 60 years. In older patients, however, idiopathic degeneration of the conduction pathways may be the major cause of RBBB. In the Reykjavik Study, hypertension had a strong association with RBBB in women younger than 60 years, whereas age had the strongest association in women older than 60 years.⁵ Age can explain the presence of RBBB in the case patient, but it does not explain the sudden change from an irregular rhythm (atrial fibrillation) to a regular slow rhythm (a junctional rhythm).

MECHANISMS OF DIGOXIN-INDUCED ARRHYTHMIAS

Digoxin is one of the oldest cardiac medications. Although it does not affect mortality, it is used to

improve symptoms of heart failure.⁶ Many patients with heart failure have electrolyte abnormalities and compromised renal function, which are predisposing factors for digoxin toxicity. Toxic levels of digoxin can induce cardiac arrhythmias; other manifestations of digoxin toxicity include nausea, vomiting, headache, fatigue, blurred vision, and altered color perception.

To better understand how digoxin induces arrhythmias, it is helpful to examine the mechanism of action and the effect digoxin has on the conduction system. Digoxin has 2 actions on the heart: a direct action of inhibiting the Na⁺, K⁺-ATPase pump (sodium pump), and an indirect action via vagal stimulation.

At the SA node, digoxin slows the heart rate mainly through vagal stimulation.^{1,7} In the atrial tissue, at therapeutic levels, digoxin has no effects, but at toxic levels, it increases automaticity and decreases conduction. At the AV junction, at low levels, digoxin slows AV nodal conduction by vagal stimulation. At toxic levels, it appears to act through its direct effects on the sodium pump to prolong the AV nodal refractory period. This direct effect at toxic levels leads not only to advanced AV nodal block, but it also enhances automaticity of the junctional tissue. The junctional tissue may begin to discharge at an increasing frequency and progress to a junctional tachycardia, recognized as paradoxical regularization of the heart rate in a patient with atrial fibrillation.^{1,7}

In the His-Purkinje system, digoxin increases automaticity and decreases the excitability of the system by its direct action on the sodium pump. This effect is seen especially in elderly patients with heart disease, in which ischemia, muscle stretch, and other factors increase Purkinje fiber automaticity. This direct action in the presence of heart disease may lead to digoxin-induced junctional and ventricular arrhythmias.

On the ventricular myocardium, digoxin has no effect at low levels, but at high levels, it causes shortening of the effective refractory period; this shortening may not be uniform throughout the ventricle, leading to toxic arrhythmias.⁸

The most common digoxin-induced arrhythmia is premature ventricular ectopy, which may be of any morphology—unifocal or multifocal, and with varying coupling intervals. If these arrhythmias do not heighten clinical suspicion and more digoxin is administered, arrhythmias that are more serious may develop. The common arrhythmias induced by digoxin are summarized in **Table 2**.

Many factors predispose to digoxin toxicity, including renal insufficiency, hypothyroidism, electrolyte abnormalities (hypokalemia, hyperkalemia, and hypercalcemia^{4,8}), the presence of structural heart disease,

Table 2. Arrhythmias Commonly Caused by Digoxin Toxicity

Enhanced automaticity (ectopic rhythm):
Atrial tachycardia with block
Nonparoxysmal junctional tachycardia
Ventricular tachycardia, fibrillation
Bidirectional tachycardia
Atrial flutter, fibrillation
Depressed conduction
Sinoatrial block or atrioventricular exit block in association with:
Sinus rhythm
Atrial tachycardia
Atrial flutter
Junctional rhythm
Ventricular tachycardia
Enhanced automaticity (ectopic rhythm) with depressed conduction:
Junctional rhythm
Ventricular tachycardia with depressed atrioventricular conduction

and interaction with other medications, including amiodarone,⁹ verapamil,¹⁰ and quinidine.¹¹ The case patient was not currently on any of these medications, her electrolyte levels were normal, and her thyroid-stimulating hormone level was normal. She did exhibit worsening renal function; this can be attributed to contrast-induced acute renal failure based on the time frame in relation to her recent computed tomography scan with contrast, the absence of other predisposing factors, and her normal kidney ultrasound. This worsening in renal function is the most likely cause of her digoxin toxicity.

CLINICAL COURSE OF CASE PATIENT

The patient was admitted to the hospital under telemetry observation. Her digoxin was stopped, and her electrolytes were frequently checked. Her kidney function improved after hydration, and her digoxin level came down to normal levels within 3 days of stopping the medication.

HP

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(continued on page 40)

(from page 25)

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