ELECTROCARDIOGRAM UNKNOWNS

Hyperkalemia-induced escape capture bigeminy

Janaki Rami Reddy Manne, MD, Jalaj Garg, MD, FACC, FESC

From the Division of Cardiology, Cardiac Arrhythmia Service, Medical College of Wisconsin, Milwaukee, Wisconsin.

Introduction

Escape-capture bigeminy can be an early sign of sick sinus node manifesting as sinoatrial exit block. Medications such as digoxin, beta-blockers, and calcium-channel blockers have been associated with escape-capture bigeminy but rarely associated with hyperkalemia. We demonstrate a case of complete resolution of the electrocardiogram (ECG) abnormalities following hyperkalemia treatment and normalization of serum potassium levels. A quick search for reversible cause and work-up is essential to avoid unnecessary delays in the recognition and treatment of the catastrophic noncardiac clinical conditions associated with this ECG finding.

Case

A 66-year-old man with a history of hypertension, insulin-dependent type II diabetes, and bladder cancer status post radical cystectomy with an open ileal conduit urinary diversion, chronic kidney disease stage III presented with generalized weakness, dizziness, and presyncope. He was afebrile, with a blood pressure of 102/62 mm Hg, heart rate 36/min, respiratory rate 14/min, and pulse oximetry 100% on room air. His physical examination was unremarkable. His home medications included amlodipine 10 mg, lisinopril 40 mg, chlorthalidone 25 mg, insulin 70/30, and atorvastatin 20 mg. Laboratory data revealed sodium 133 mEq/L (136–145), chloride 112 mEq/L (98–107), potassium 7 mEq/L (3.5–4.5), bicarbonate 15 mEq/L (24–26), blood urea nitrogen 79 mg/dL (7–21), creatinine 2.88 mg/dL (0.67–1.17). His 12-lead ECG at presentation is shown in Figure 1.

What would you do next?

(A) Place an urgent temporary transvenous pacemaker
(B) Electrophysiology study
(C) Treat hyperkalemia
(D) Place a permanent dual-chamber pacemaker

KEY TEACHING POINTS

- Escape-capture bigeminy can be an early sign of sick sinus node manifesting as sinoatrial exit block. Medications such as digoxin, beta-blockers, and calcium-channel blockers have been associated with escape-capture bigeminy but rarely associated with hyperkalemia.
- Complete resolution of the electrocardiogram (ECG) abnormalities was demonstrated with hyperkalemia treatment and normalization of serum potassium levels.
- A quick search for reversible cause and work-up is essential to avoid unnecessary delays in the recognition and treatment of the catastrophic noncardiac clinical conditions associated with this ECG finding.

What to do next?

C. Treat hyperkalemia

Diagnosis: hyperkalemia-induced escape-capture bigeminy

Commentary

The 12-lead ECG at presentation demonstrates repetitive group beating in a bigeminy fashion. Bigeminal rhythm due to a ventricular escape beat followed by a sinus conducted beat is very uncommon. Escape-capture bigeminy is characterized by repetitive group beating of escape beat followed by sinus conducted beat.[1] The 12-lead ECG shows sinus bradycardia at a rate of 36/min with a P-P interval of 1600 ms. Each P wave (best seen in lead V_1) is followed by a QRS complex with a PR interval of 200 ms. A ventricular escape or a junctional escape rhythm with intraventricular conduction delay from hyperkalemia is seen interrupting each sinus cycle at a constant escape interval of 1180 ms (Figure 2). The ventricular or junctional escape beat is followed by a regular sinus beat at intervals of 420 ms (a pattern that repeats itself) (Figure 2). In escape-capture bigeminy, the escape rhythm can originate from the atrioventricular junction or the ventricle

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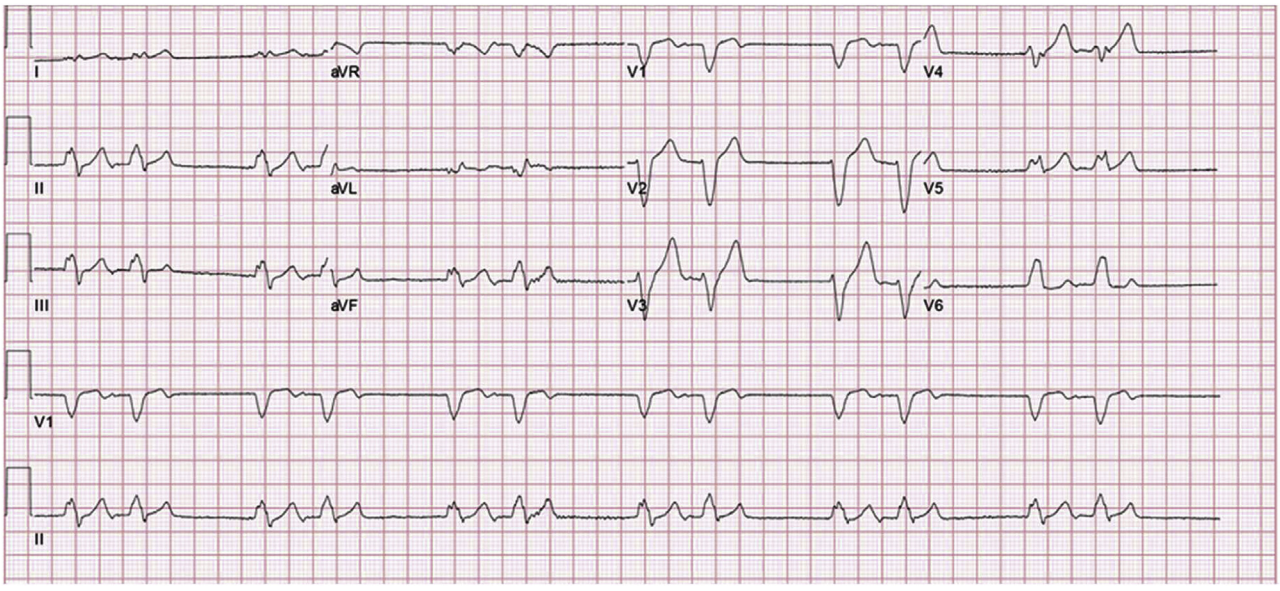

Figure 1 Twelve-lead electrocardiogram at presentation.

owing to intermittent block of the sinus impulse at the level of either the sinus or atrioventricular node. For the emergence of escape-capture rhythm, the effective cycle length of the primary pacemaker must exceed the escape interval plus the refractory period following the escape complex. The escape beat does not reset the sinus node owing to either a retrograde conduction block or entrance block at the sinus node level.

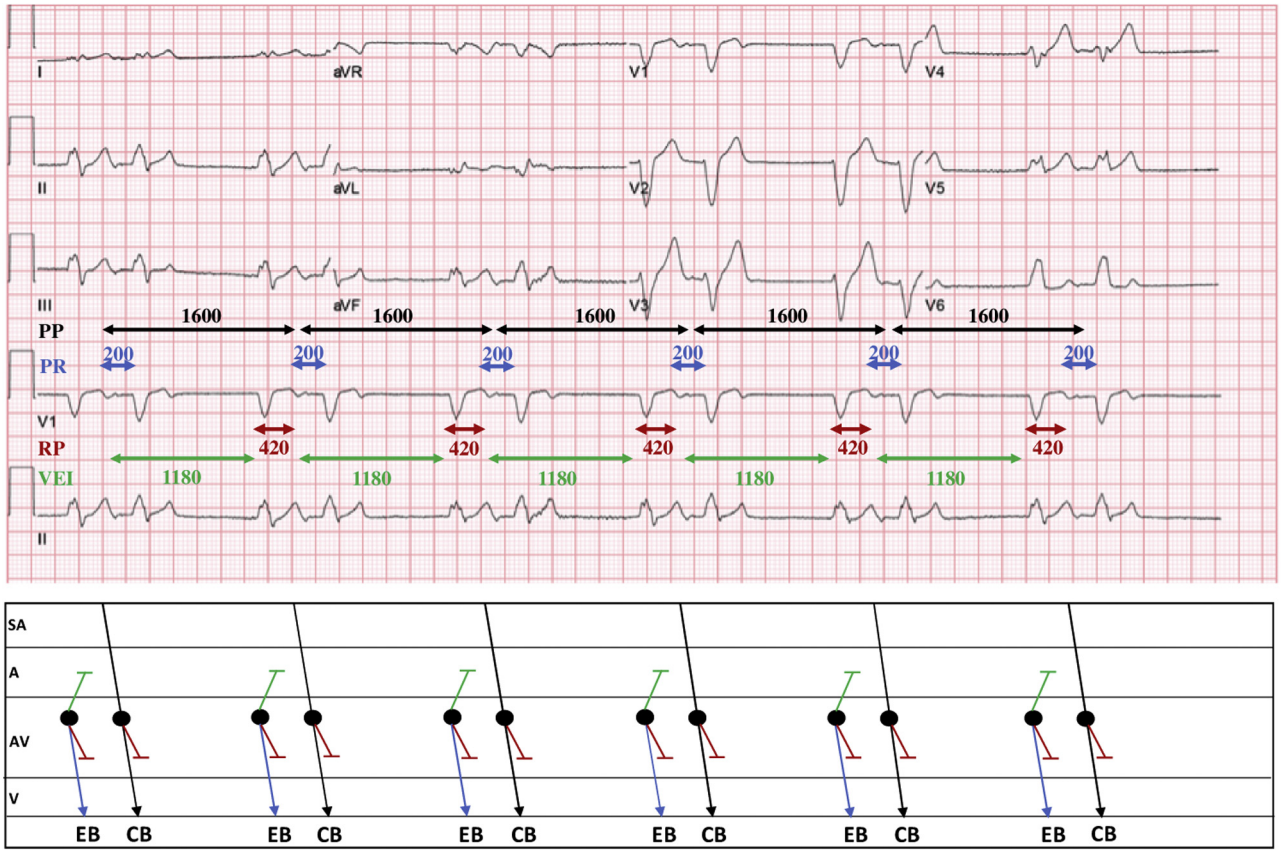

Figure 2 Twelve-lead electrocardiogram with a corresponding ladder diagram demonstrating escape capture bigeminy. There is no retrograde atrial conduction during the left bundle branch escape beat (EB). Capture beat (CB) occurs with the constant R-R interval of 620 ms (RP + PR or 420 + 200 ms) with left bundle branch block (similar to EB); the pattern that repeats itself. (The cycle lengths for the PP, PR, and RP intervals are denoted in milliseconds). VEI = ventricular escape interval.

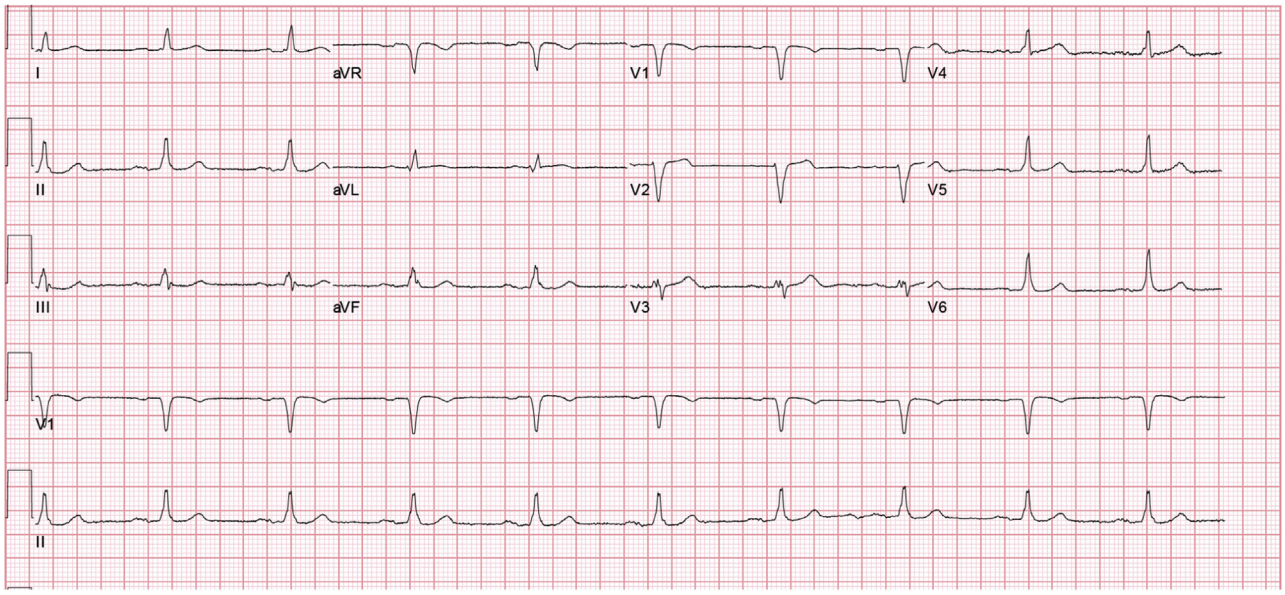

Figure 3 Repeat 12-lead electrocardiogram following treatment of hyperkalemia.

The escape-capture rhythm seen in our patient most likely indicates a sinoatrial block, allowing for a focus below the sinoatrial node to escape and the emergence of escape-capture bigeminy. However, a junctional or ventricular escape beat with retrograde conduction to the atria and subsequent anterograde conduction cannot be completely ruled out (echo beat). Escape-capture bigeminy can be an early sign of sick sinus node manifesting as sinoatrial exit block.[1] Medications such as digoxin, beta-blockers, and calcium-channel blockers have been associated with escape-capture bigeminy[2,3] but rarely associated with hyperkalemia. Our patient was not on any nodal blocking agents. Intrinsic sinus node dysfunction cannot be excluded entirely in our case; however, complete resolution of the ECG abnormalities with the hyperkalemia treatment and normalization of serum potassium levels suggests hyperkalemia as the most likely etiology.

In hyperkalemia, the increased extracellular potassium initially decreases the resting membrane potential and shortens the action potential duration in all cardiac tissues by increasing the velocity of the phase 3 repolarization (increased Ikr potassium efflux).[2] The ECG findings seen at this stage include peaking of the T waves, shortening of the QT interval, and ST-segment depression. As the resting membrane potential approaches threshold value, the myocardial cell becomes less excitable owing to reduced sodium influx, resulting in a decrease in the rate of rise and voltage of phase 0 of the action potential.[2] This leads to decreased conduction velocity across the myocardial tissue manifesting as reduced amplitude and duration of the P wave, prolonged P-R interval, widening of QRS complex with the eventual appearance of the sine wave pattern, and, subsequently, asystole.

The QRS widening demonstrated in hyperkalemia may be due to a localized block in the His-Purkinje system or diffuse conduction delay in the ventricular myocardium.[4] This can manifest in the form of hyperkalemia-induced bundle branch block, hemifascicular block, second-degree atrioventricular block, and complete heart block in the absence of any preexisting conduction system disease.[5]

Patient outcome

The patient was treated with intravenous calcium gluconate, insulin, dextrose, and oral kayexalate, which resolved the hyperkalemia and the escape-capture bigeminy (Figure 3). Lisinopril was discontinued, and he was placed on oral bicarbonate before discharge. He remained asymptomatic on follow-up, and a 1-month mobile cardiac telemetry monitor demonstrated no brady/tachyarrhythmias or conduction blocks.

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