

CASE REPORT

A case of atenolol-induced sinus node dysfunction presenting as escape-capture bigeminy

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Abstract

Medications that are routinely used in clinical practice to treat hypertension such as beta blockers or non-dihydropyridine calcium channel blockers can depress sinus node function, resulting in symptoms and electrocardiographic changes consistent with sinus node dysfunction. In some patients, medications are potentially a reversible cause of apparent sinus node dysfunction. We report a patient who was taking atenolol for hypertension presented with recurrent near syncopal spells due to atenolol-induced sinus node dysfunction manifested in the form of an interesting and rare arrhythmia known as Escape-capture bigeminy.

INTRODUCTION

Escape-capture bigeminy is an interesting arrhythmia characterized by repetitive group beating of escape beat followed by a sinus capture beat. This is a very uncommon arrhythmia since it occurs only when the sinus cycle length is longer than the escape cycle interval plus the refractory period following the escape complex.

CASE REPORT

A 65-year-old Caucasian male with a history of poorly controlled hypertension and hyperlipidemia presented to the emergency room for evaluation of near syncope. He complained 1 week history of dizziness and near syncopal events but denied any chest pain, shortness of breath or palpitations. He was taking 10 mg of amlodipine, 50 mg of atenolol and 81 mg of aspirin on a daily basis. During his recent office visit, he was found to have an elevated blood pressure of 165/90 mmHg and the atenolol dose was increased to 75 mg for better blood pressure control.

On admission, he was registering a temperature of 98.4°F, a blood pressure of 80/40 mmHg without orthostatic hypotension, a pulse rate of 40 beats/min, respiratory rate of 16/min and oxygen saturation of 96% on room air. Physical examination was unremarkable. During his physical exam, he experienced another near syncopal event. Electrocardiogram obtained showed repetitive group beating in a bigeminal pattern. The 12-lead ECG (Fig. 1) shows sinus bradycardia at a rate of 36 beats/min with a P–P interval of 1840 ms. Each P wave is followed by a QRS complex with a PR interval of 200 ms. The P waves (green arrows) are best seen in lead II. A junctional escape rhythm is seen interrupting each sinus cycle at a constant escape interval of 1360 ms. The junctional escape beats are followed by regular sinus beats at intervals of 480 ms. Laboratory data including thyroid stimulating hormone, electrolytes and serial cardiac markers (troponin I) were within normal range. Transthoracic echocardiogram did not reveal any abnormalities. He had no signs or symptoms of extra cardiac sarcoidosis and did not meet the criteria for advanced cardiac imaging to look for

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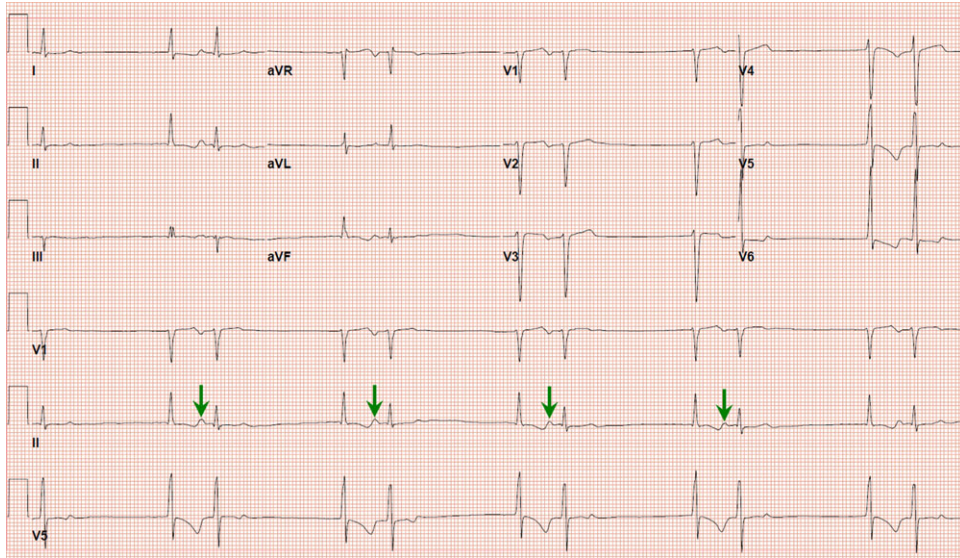


Figure 1: Admission ECG showing marked sinus bradycardia and repetitive group beating of junctional escape beat followed by a sinus beat in a bigeminal fashion. The upright sinus P waves (green arrows) are best seen in lead II.

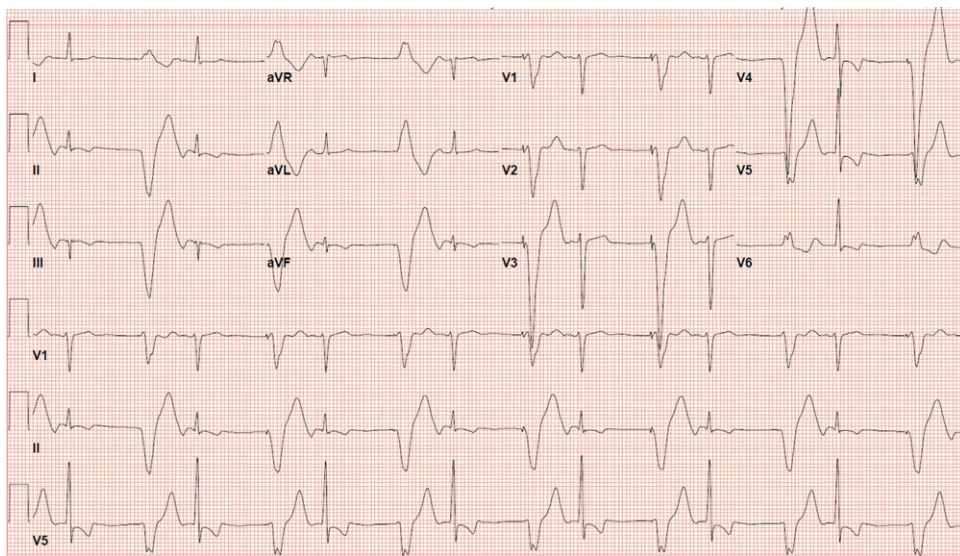


Figure 2: ECG after placing a temporary pacemaker showing sinus bradycardia and the junctional rhythm is replaced by the ventricular-paced rhythm. Alternating ventricular-paced complexes and sinus complexes are seen in a pattern of bigeminy.

cardiac sarcoidosis. His recent coronary angiogram a month ago showed normal coronaries.

Since beta blockers can suppress the sinus node function, we discontinued atenolol. The patient underwent temporary transvenous pacemaker placement (demand pacing at 80 beats/minute) because of symptomatic bradycardia and hemodynamic instability (Fig. 2).

After 36 hours, his intrinsic sinus rate significantly improved and the temporary pacemaker was removed. The junctional escape rhythm and the escape-capture bigeminy also disappeared with improvement of the sinus rate (Fig. 3). Telemetry monitoring for the next 48 hours was uneventful and he was discharged on mobile cardiac outpatient telemetry. He was also started on 10 mg of Lisinopril before discharge. On 1-month follow-up, he was asymptomatic with a blood pressure of 124/

68 mmHg and no abnormal rhythms were recorded on the cardiac monitor. Atenolol-induced suppression of sinus automaticity or sinoatrial exit block is the most likely cause of dizziness and near syncope in our patient. As calcium channel blockers can also suppress the sinus node function, one cannot rule out the role of amlodipine in the contribution of the patient symptoms without rechallengeing him with atenolol in the absence of amlodipine. However, he did not report any symptoms thus far while he continued to take amlodipine for hypertension.

DISCUSSION

Escape-capture bigeminy refers to a bigeminal rhythm where an escape beat (atrial, junctional or ventricular) is followed by a sinus capture beat. It is also known as 'Reversed bigeminy' or

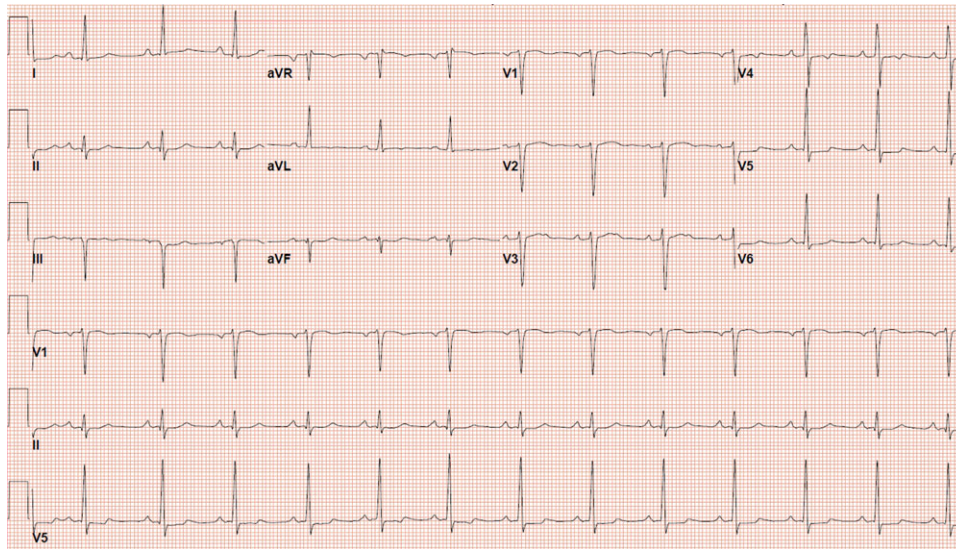


Figure 3: ECG before discharge showing improved sinus rate, resolution of junctional escape rhythm and escape-capture bigeminy.

'Pseudo-reciprocal rhythm'. It resembles reciprocal rhythm in having a P wave sandwiched between two QRS complexes. However it differs from a true reciprocal rhythm by the presence of a normal P wave and absence of retrograde activation. The term Escape-capture bigeminy was coined by Bradley and Marriott [1] in preference to pseudo-reciprocal rhythm, where they described a case of 2:1 sinoatrial block manifested as escape-capture bigeminy. Its occurrence has also been described in association with marked sinus bradycardia, junctional escape rhythm, idioventricular and pacemaker induced ventricular escape rhythm [2]. Escape-capture bigeminy emerges when the effective sinus cycle length exceeds the sum of the escape cycle interval and the refractory period following the escape beat. This requires an intermittent block of the sinus impulses at either the sinus or A-V nodal level [3]. Hence the escape beat can originate from the atrium, A-V junction or the ventricle. Junctional or ventricular escape beats with retrograde conduction to the atria with reciprocal anterograde conduction must be ruled out. In escape-capture bigeminy of junctional origin, the junctional rate is faster than the sinus rate as seen in our patient. The escape interval is controlled by the intrinsic junctional rate. The escape beat does not alter the sinus cycle length or reset the slower sinus node either because the escape complex is blocked from retrograde conduction or because of entrance block at the sinus node level. When the sinus cycle length decreases or in other words when the sinus rate increases sufficiently, the escape rhythm and the escape-capture bigeminy will disappear.

Although the sinus cycle length observed in our patient is 1840 ms, the actual sinus cycle length is probably twice as rapid; 920 ms with 2:1 sinoatrial block resulting in junctional escape-capture bigeminy. Sinoatrial exit block results from the failure of generation or propagation of sinus node impulses to the atria. It is characterized by the absence of P waves on the surface electrocardiogram. There are no established criteria available for the diagnosis of sinus node dysfunction. The typical ECG findings seen during symptomatic presentation include sinus bradycardia; sinus pause/arrest, alternating bradycardia and atrial tachyarrhythmias, sinoatrial exit block. The correlation between the patient symptoms and the underlying rhythm at the time of symptomatic presentation is critically important to establish the diagnosis. In some patients,

however, additional ECG monitoring and work up may be required but sinus node disease should not be diagnosed until a careful review is performed to identify and treat any potentially reversible causes (e.g. medications, myocardial ischemia and hypothyroidism) of apparent sinus node dysfunction. Medications such as beta blockers, digitalis, calcium channel blockers and antiarrhythmic drugs can suppress sinus node function. They have also been implicated in the etiology of escape-capture bigeminy [4].

In conclusion, escape-capture bigeminy may suggest a drug induced rhythm but sometimes it may be the only ECG manifestation of sinus node dysfunction or sinoatrial exit block. It is clinically important to recognize this dysrhythmia, as it needs careful monitoring and evaluation. Sometimes discontinuation of the non-essential medications that can depress sinus node function is required to restore normal sinus rhythm. However, placement of a permanent pacemaker is indicated for patients with symptomatic sinus dysfunction that results from required drug therapy of a type and dose for which there is no acceptable alternative available [5] (Level 1; Class C evidence).

CONFLICT OF INTEREST STATEMENT

None declared.

FUNDING

None.

ETHICAL APPROVAL

Not applicable.

CONSENT

Not applicable.

GUARANTOR

Janaki Rami Reddy Manne.

REFERENCES

1. Bradley SM, Marriott HJ. Escape-capture bigeminy; report of a case of A-V dissociation initiated by 2:1 S-A block with resulting bigeminal rhythm. *Am J Cardiol* 1958;1:640.
2. Fisch C. *Electrocardiography of Arrhythmias*. Philadelphia: Lea & Febiger, 1990.
3. Schamroth L, Dubb A. Escape-capture bigeminy. Mechanisms in S-A block, A-V block, and reversed reciprocal rhythm. *Br Heart J* 1965;27:667-9.
4. Monir G, Dreifus LS, Gursoy AS, Kutalek SP. Escape capture bigeminy: a manifestation of sinoatrial conduction block. *J Electrocardiol* 1999;32:51.
5. Epstein AE, DiMarco JP, Ellenbogen KA, Estes NM, Freedman RA, Gettes LS, et al. ACC/AHA/HRS 2008 Guidelines for device-based therapy of cardiac rhythm abnormalities. *Heart Rhythm* 2008;5:e1-62.