Concealed His Extrasystoles: A Masquerader of AV Block

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Abstract

Concealed His extrasystoles are a well-known masquerader of AV block. It is therefore necessary to consider pseudo AV block in patients who develop unexpected AV block without a clear etiology. In this report, we present a case of a 46-year-old female who was found to have pseudo-Mobitz II AV block secondary to His extrasystoles diagnosed on surface ECG without requiring invasive electrophysiology study. We also demonstrated in this patient that flecainide can be an effective treatment for His extrasystoles.

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Introduction:

Concealed His extrasystoles are a well-known masquerader of AV block. It is therefore necessary to consider pseudo AV block in patients who develop unexpected AV block without a clear etiology. In this report, we present a case of a 46-year-old female who was found to have pseudo-Mobitz II AV block secondary to

His extrasystoles diagnosed on surface ECG without requiring invasive electrophysiology study. We also demonstrated in this patient that flecainide can be an effective treatment for His extrasystoles.

Case Report:

We present a case of a 46-year-old female with a history of anxiety and hypothyroidism who was referred to our clinic for possible pacemaker implantation. Her baseline 12-lead ECG is shown in Figure 1. She wore an ambulatory ECG monitor for 2 weeks after complaints of palpitation during which time 156 episodes of second-degree Mobitz II AV block were observed (Figure 2). Lyme titer was negative. Transthoracic echocardiogram revealed normal LV size and function without significant valvular heart disease. Cardiac MRI showed normal biventricular size and function with no late gadolinium enhancement. She had good exercise capacity (9 METS) with no evidence of ischemia on treadmill exercise stress test. During stage I of the Bruce protocol, there was a single non-conducted P wave following the 10^{th} QRS complex with fixed PR and PP intervals consistent with Mobitz II AV block (Figure 3). Sinus tachycardia with frequent likely junctional extrasystoles with a typical right bundle branch block (RBBB) configuration were also present. Closely following the blocked P wave was a small negative deflection consistent with a retrograde P-wave. The P-wave is narrow and inverted in the inferior leads consistent with retrograde septal activation as would be expected to occur in association with a junctional extrasystole. While this could have been an opportunistic premature atrial extrasystole, the more likely explanation is that a junctional extrasystole conducted retrogradely through the AV node to the atrium and blocked antegradely in the His Purkinje system (Figure 4). The timing of the extrasystole would have had to occur during inscription of the blocked antegrade P wave and engaged the AV node retrogradely causing the oncoming antegrade P wave to block in the AV node. Furthermore, Mobitz II block is typically secondary to conduction disease below the AV node and tends to get worse at a faster heart rate, which was not observed here. All findings were supportive of pseudo AV block secondary to His extrasystoles. She was started on Flecainide which was up titrated to 100 mg twice daily. A repeat ambulatory ECG recording at 5 months follow-up showed normal sinus rhythm with no AV block. A repeat treadmill exercise stress test revealed a peak heart of 153 bpm without evidence of ischemia or AV block.

Discussion:

Concealed His extrasystoles mimicking AV block was first described by Langendorf and Mehlman in 1947[1]. The mechanism was postulated to be secondary to His extrasystoles that fail to conduct to the ventricle but partially conduct into the AV junction. This was later confirmed in an electrophysiology study (EPS) in 1970 by Rosen et al[2]. AV block secondary to His extrasystoles has been termed pseudo AV block. This phenomenon was well documented in the 1970s[3-5]. There have been few scattered case reports since then. In this case report, we present an otherwise healthy 46-year-old female who was found to have 156 episodes of second-degree Mobitz II AV block on ambulatory monitoring for palpitations. Evaluation for reversible causes with Lyme titers, transthoracic echocardiogram, cardiac MRI, and treadmill exercise stress test were unremarkable. However, ECG during her stress test provided insight into the cause of her AV block. ECG in Figure 3 showed sinus tachycardia, junctional bigeminy with RBBB, and a blocked sinus P wave that was closely coupled to a small negative deflection consistent with an atrial depolarization from a His extrasystole that conducted retrogradely to the atrium but failed to conduct to the ventricle and partially conducted into the AV node resulting in a pseudo AV block. This occurrence was supported by the observation of frequent junctional bigeminy that conducted with RBBB. At a shorter coupling interval, a His extrasystoles likely blocked antegrade in the His Purkinje system and failed to depolarize the ventricles. In addition, it also conducted retrograde into the slow AV node pathway resulting in retrograde atrial activation and into the fast AV node pathway causing the preceding sinus beat to block in the AV node (pseudo-AV block) as illustrated in Figure 4.

In 1973, Narula and Eugster et al. suggested that AV block from His extrasystoles could be a sign of conduction disease distal to the His bundle in some patients indicating the need for a pacemaker[6-7]. However, in 1976, Booner and Zipes showed no evidence of distal conduction disease in some patients with pseudo AV block[9]. While we cannot rule out with absolute certainty the presence of distal conduction system disease in

this case without an electrophysiology study, the likelihood of distal disease islow for the following reasons. At baseline, this patient had normal PR interval and QRS duration. Second, she had no AV block at peak exercise (153 bpm). These findings along with her frequent ectopy were supportive of pseudo AV block secondary to concealed His extrasystoles as opposed to distal conduction system disease. After a lengthy conversion with the patient, electrophysiology study was deferred and Flecainide was started. While there have been no reported case studies of using Class IC antiarrhythmics to treat His extrasystoles, we chose Flecainide based on the results of previous studies using other class I antiarrhythmic agents as shown in Table 1. Five months after initiation of Flecainide, a repeat ambulatory ECG recording showed no AV block. Patch monitor showed rare PACs and PVCs. A repeat treadmill stress test showed rare junctional complexes without AV block.

The findings in this patient underscore the fact that concealed His extrasystoles can be diagnosed from careful analysis of the surface electrocardiogram, and Flecainide is an effective treatment for pseudo AV block from concealed His extrasystoles. Treatment of pseudo AV block from His extrasystoles with class I antiarrhythmic agents is summarized in Table 1.

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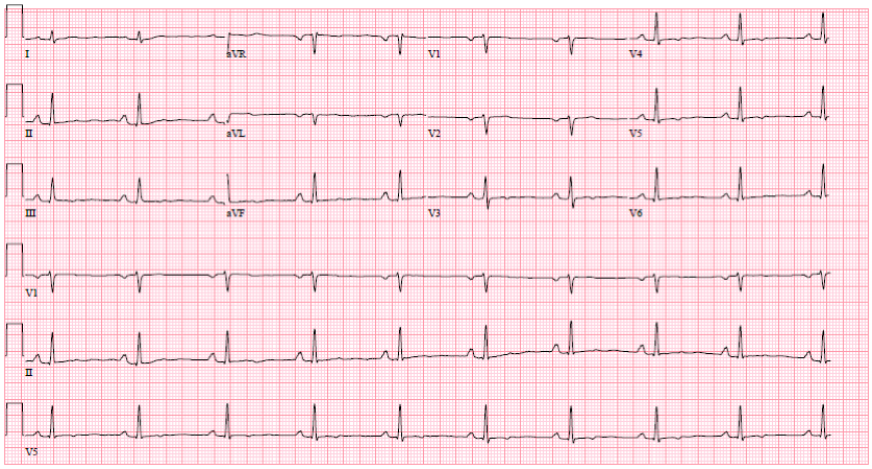

Figure 1 : Baseline ECG shows sinus bradycardia

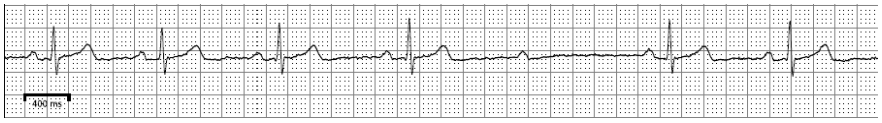

Figure 2: A rhythm strip from ambulatory monitor. The PP and PR intervals are constant before and after the blocked P wave consistent with Mobitz II AV block.

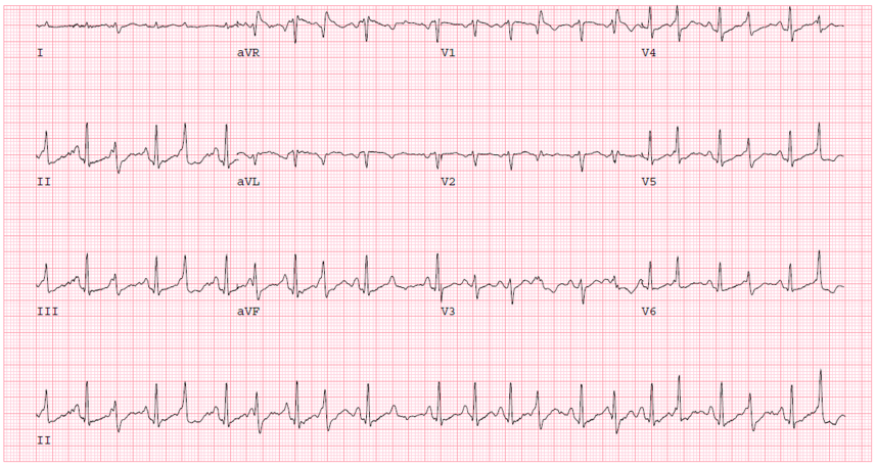

Figure 3: ECG during treadmill exercise stress test(Stage I Bruce protocol). Sinus tachycardia with frequent junctional or ventricular premature complexes and Mobitz II AV block are present.

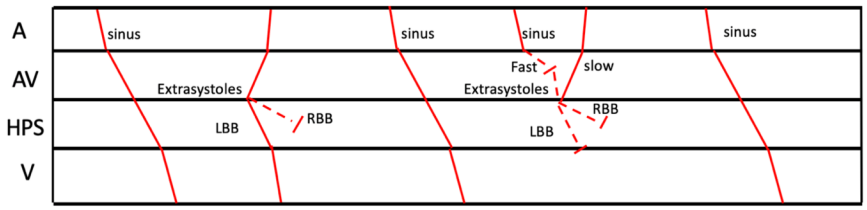

Figure 4: Ladder diagram illustrates our proposed mechanism for intermittent RBBB and AV block seen on ECG. A His extrasystoles conducts retrogradely through the AV node to the atrium but blocks antegradely in the RBB will give rise to a QRS with RBBB. A shorter coupled His extrasystoles conducts retrograde into the slow AV node pathway to the atrium and into the fast AV node pathway causing the preceding sinus beat to block in the AV node will give rise to an inverted P wave without QRS mimicking AV block.

Authors	**Year**	**Findings**
Bonner and Zipes[8]	1976	Lidocaine eliminated His extrasystoles
Dhurandhar, Valen, and Phillips[9]	1976	Quinidine sulphate 200 mg four times a day completely suppressed His extrasys
Castellanos et al[10]	1977	Xylocaine markedly reduced fascicular extrasystoles
Khan et al[11]	2011	Procainamide suppressed His extrasystoles

Table 1 : Case studies on treatment of pseudo AV block from His extrasystoles