CASE REPORT

Progressive interatrial block associated with atrial fibrillation in a patient with hypertrophic cardiomyopathy

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Progressive interatrial block is a clinically significant condition that has previously been reported in various patient populations. It is a manifestation of progressive fibrosis affecting the Bachmann region. This report presents a case of progressive interatrial block associated with atrial fibrillation in the context of hypertrophic cardiomyopathy.

KEYWORDS

atrial fibrillation, Bayés' syndrome, hypertrophic cardiomyopathy, interatrial block

Interatrial block (IAB) is a well-described electrocardiographic phenomenon that entails a delayed conduction between the two atria during atrial systole. IAB has further been classified into partial IAB (pIAB), which is seen on the surface electrocardiogram (ECG) as P waves of 120 ms or more, or advanced IAB (aIAB) in which P waves are also prolonged but with a biphasic morphology depicting a negative terminal portion in the inferior leads (II, III, and aVF; Bayes de Luna et al., 2012). This pattern arises owing to a delay of conduction at the Bachmann region resulting in retrograde, caudocranial conduction to the left atrium (LA) through the coronary sinus musculature (Cosio et al., 2004). It has previously been demonstrated through serial ECGs that normal P waves and pIAB can progress to aIAB over time (Ariyaratne & Spodick, 2006; Enriquez et al., 2015). IAB has been associated with increased incidence of atrial fibrillation (AF), likely owing to delayed, heterogeneous activation of the LA (Bayes de Luna et al., 2012). Hypertrophic cardiomyopathy (HCM) is defined by increased left ventricular wall thickness or mass without hemodynamic stressors that could account for these changes (Elliott et al., 2008). The following case is a description of progressing aIAB associated with AF in a patient with HCM.

1 | CASE PRESENTATION

A 72-year-old male with HCM treated with bisoprolol 2.5 mg daily, presented to clinic for regular follow-up. The patient showed evidence of HCM on a baseline ECG in 2010 which displayed features of asymmetrical septal hypertrophy through dagger-like Q waves in lateral

and inferior leads (Fig. 1). The diagnosis was confirmed by echocardiogram and cardiac magnetic resonance imaging (MRI). The baseline ECG also displayed biphasic P waves with a duration >120 ms and a negative terminal component (Fig. 1) indicating aIAB (Bayes de Luna et al., 2012).

Serial ECGs from 2010 until 2016 have shown progressive lengthening of the P-wave duration (from 150 ms in 2010 to 187 ms in 2016). A comparison of ECGs in 2010 and 2016 reveals the evolution of aIAB through morphological changes in the negative component of the P wave. This includes an increase in duration (from 94 ms in 2010 to 132 ms in 2016) and amplitude (from 0.094 mV in 2010 to 0.136 mV in 2016) of the specific negative component of the P wave measured in the inferior leads (Fig. 2). Furthermore, ECG testing in 2015 documented the presence of new-onset AF that was successfully cardioverted back to sinus rhythm (Fig. 3, Panels A,B).

Magnetic resonance imaging and serial echocardiograms documented the progression of HCM as well. Posterior wall thickness increased from 11 mm in 2010 to 13 mm in 2012 (normal parameters: 7–11 mm). Increasing left ventricular hypertrophy was evidenced by an increase in basal septal thickness from 11 mm in 2010 to 19 mm in 2015 (normal parameters: 7–11 mm). The 2015 echocardiogram also showed the presence of systolic anterior movement of the mitral valve, a classic feature of HCM (Fig. 3C). Cardiac MRI from 2015 displayed significant LA enlargement with a diameter of 5 cm (normal parameter: 1.5–4 cm) and also exhibited predominantly septal hypertrophy of the left ventricle with significant myocardial fibrosis (Fig. 3D).

FIGURE 1 Baseline 12-lead ECG performed in 2010 showing evidence of partial interatrial block and hypertrophic cardiomyopathy

2 | DISCUSSION

Through a variety of imaging modalities over several years, we observed the progression of aIAB and onset of AF in a patient with advancing HCM. The progression of aIAB was detected on ECG through the prolongation of the P wave duration and an increase in length and duration of the negative terminal component of the P wave in the inferior leads. This pattern indicates a higher degree of delayed conduction through the Bachmann region resulting in increased caudocranial activation of the LA through an alternative myocardial pathway; usually the musculature of the coronary sinus (Cosio et al., 2004).

It has previously been established that atrial remodeling predisposes the heart to both conduction delay and subsequently arrhythmias (Ariyaratnam & Spodick, 2006). In this patient, a moderate increase in left ventricular outflow tract pressure produced systolic anterior

movement of the mitral valve, leading to valvular insufficiency and subsequent increase in LA diameter.

A previous study by Holmqvist et al. (2007) including 65 patients with HCM has found that the P-wave duration of patients with IAB (transformed into orthogonal lead data) was significantly longer than in the control group (149 ± 22 vs. 130 ± 16 ms, $p < .0001$).

Supraventricular tachycardias are frequently associated with aIAB (mostly AF), and their co-occurrence has been coined Bayés' syndrome (Bayes de Luna et al., 2012). Furthermore, it has been shown that a subgroup of patients satisfying aIAB criteria with a P-wave duration >140 ms had a significantly higher rate of AF recurrence following pulmonary vein isolation as a therapy than those aIAB patients with P-wave durations ranging from 120–140 ms (Caldwell et al., 2014). This demonstrated that progression of aIAB could increase the risk of AF and thus the onset of Bayés' Syndrome.

FIGURE 2 Electrocardiographic progression of P wave. Note increased P-wave duration and morphology change from 2010 to 2016 in the inferior leads (II, III, and aVF)

This case represents the progression of aLAB as part of Bayés' syndrome occurring in the context of HCM. Bayés' syndrome has been demonstrated in numerous patient populations, including hemodialysis patients, patients with severe heart failure, and in hypertensive patients (Ali et al., 2015; Blanche et al., 2014; Francia et al., 2015; Marano, D'Amato, & Cantone, 2016). As in these scenarios, is important to identify HCM as an etiologic factor for Bayés' syndrome as these patients could benefit from early intensive cardiac monitoring

and more aggressive antiarrhythmic or anticoagulation treatment to prevent further cardiovascular complications (Alexander & Baranchuk, 2016). AF in the context of HCM can contribute to hemodynamic intolerance and facilitate negative cardiac remodeling.

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FIGURE 3 Twelve-lead ECG depicting AF prior to electrocardioversion (A). Twelve-lead ECG in sinus rhythm following electrocardioversion (B). Echocardiogram of heart performed in 2015 depicting moderate concentric hypertrophy of the left ventricle, systolic anterior movement of the mitral valve, and dilatation of the aortic root (C). Magnetic resonance imaging of the heart displaying left ventricular hypertrophy. Arrows indicate areas of fibrosis (D)

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