

Widely split P waves in a patient with atrial enlargement and prior atrial flutter catheter ablations



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Introduction

The P wave represents the electrical depolarization of the atria. A uniform-shaped P wave can reflect the conduction of depolarization through semi-specialized pathways along the atria, such as the Bachmann bundle and regions of preferential conduction in the right atrium. Different P wave morphologies following sinus node impulse formation can be caused by electromechanical abnormalities and may serve as helpful information for the diagnosis of certain cardiac conditions. Rarely, P waves can be extremely prolonged and take on a widely split morphology, the differential of which is broad and warrants thorough history-taking and comprehensive clinical work-up. Here, we report a unique case of a patient with a complex cardiac history and extremely prolonged and widely split P waves. We highlight the educational value in the clinical reasoning behind an attempt to distinguish between possible causes of such electrocardiographic findings.

Case report

A 67-year-old man with a past medical history significant for recurrent atrial flutter, coronary artery disease, mitral regurgitation, diastolic dysfunction, hypertension, and end-stage renal disease presented to the hospital with acute on chronic dyspnea. The patient had been experiencing shortness of breath both with exertion and at rest for years. He underwent 2 cavotricuspid isthmus radiofrequency ablations for atrial flutter 7 and 5 years ago (in 2015 and 2017, respectively), and had been taking amiodarone 200 mg daily. Three months prior to presentation, he had an elective stent placement to the left anterior descending artery and was switched from clopidogrel to ticagrelor 90 mg twice daily. He had experienced worsening dyspnea since. He recalled being informed by a

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KEY TEACHING POINTS

- P waves result from the electrical activation of the right and left atria sequentially through semi-specialized pathways in the Bachmann bundle zone and along regions of preferred conduction within the right atrium.
- Prolonged and widely split P waves can have multiple possible causes, eg, interatrial conduction delay from atrial enlargement or remodeling, changes secondary to radiofrequency ablation, or alteration in conductive tissue properties with medication use.
- A comprehensive clinical history and work-up with imaging over an extended period of time may aid in the differential between these causes.

cardiologist that his mitral valve was regurgitant and might require intervention. His other medications included aspirin 81 mg daily, metoprolol succinate 25 mg daily, amlodipine 5 mg daily, and evolocumab 140 mg subcutaneously every 2 weeks. Because of a history of gastrointestinal bleeding, he was not receiving oral anticoagulation therapy.

On admission to the hospital, his vital signs were within normal limits, except for an elevated blood pressure of 150/80 mm Hg. High-sensitivity troponin was 44 ng/L (normal range: 0–35 ng/L) initially, 43 ng/L on repeat, and B-type natriuretic peptide was 14,135 pg/mL (normal range: 0–100 pg/mL). A chest radiograph showed an enlarged cardiac silhouette and mild volume overload changes. An electrocardiogram (ECG) showed P waves that were prolonged in duration (~200 ms) and widely split in morphology, and a prolonged PR interval at 322 ms (Figure 1). A transthoracic echocardiogram showed a left ventricular ejection fraction of 55%, moderate-to-severe mitral regurgitation, grade 3 diastolic dysfunction, enlarged right atrium, and a markedly enlarged left atrium (end-systolic anterior-posterior dimension = 58 mm; end-systolic volume = 180 mL). A transesophageal

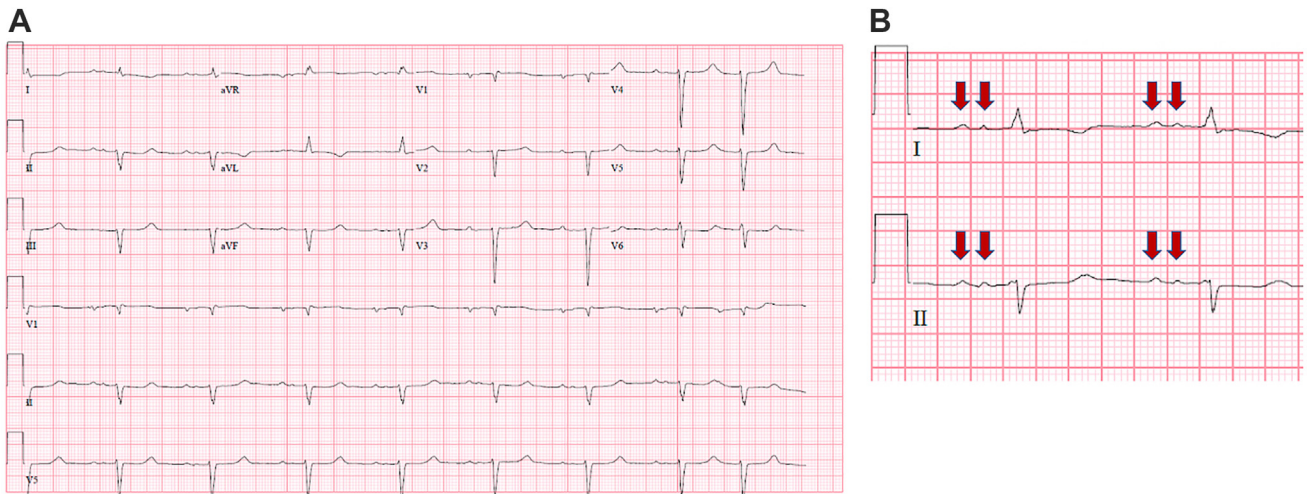


Figure 1 Twelve-lead electrocardiogram (A) with widely split P waves (~ 200 ms) best visualized in leads I and II (B) and prolonged PR interval indicating first-degree atrioventricular block.

echocardiogram found moderate mitral regurgitation (effective regurgitant orifice = 0.29 cm^2 ; regurgitant volume 41 mL).

The patient's dyspnea was attributed to a combination of mitral regurgitation, medication effect, volume overload, and diastolic heart failure. He underwent hemodialysis and was transitioned from ticagrelor back to clopidogrel. Amiodarone was reduced from 200 mg to 100 mg daily. For further symptom alleviation, the patient elected for a transcatheter mitral valve repair. At an office visit 3 months later, the patient had participated in cardiac rehabilitation and was significantly less dyspneic. However, an ECG showed the same P wave morphology.

A comprehensive review of medical records was performed to further assess for the cause of this abnormal ECG finding. ECGs showing sinus rhythm prior to the patient's second catheter ablation 5 years ago and in the year after the procedure (2018) were examined. Widely split P waves and PR interval prolongation were already present, but not as pronounced, and did not change during this time period (Figure 2). Since 5 years ago, the patient's metoprolol dose had remained the same, except for an uptitration to 100 mg daily for 1 month because of elevated blood pressure. Unfortunately, intraoperative data from the ablation procedures, both performed at outside hospitals, were unavailable. The patient had not undergone ambulatory rhythm monitoring or advanced cardiac imaging studies such as magnetic resonance imaging of the heart. A transthoracic echocardiogram 10 years ago (2012) showed trivial mitral regurgitation, a moderately enlarged left atrium (end-systolic anterior-posterior dimension = 49 mm; end-systolic volume = 106 mL) but a normal-sized right atrium.

Discussion

P waves result from the electrical activation of the right and left atria sequentially through semi-specialized pathways in the Bachmann bundle zone and along regions of preferred

conduction within the right atrium. Prolonged P waves indicate either abnormal lengthening of these pathways, frequently seen with atrial enlargement, or intra-atrial conduction disturbances, of which bimodal, widely split P waves are a frequent manifestation.¹

In this patient, the progression of biatrial enlargement temporally correlated with the lengthening of P wave duration. The uniquely wide splitting of P waves, on the other hand, could potentially have been iatrogenic, resulting from prior radiofrequency ablations, which may alter the origins of atrial impulse formation and redirect preferential conduction within the atria. Missing ECG data limited our ability to assess the evolution of P wave abnormality surrounding the patient's first atrial flutter catheter ablation, yet comparing ECGs prior to and shortly after the second ablation, the P waves were relatively unchanged in duration and morphology, and only became more prolonged and widely split in the following years. While electrophysiology study and biatrial activation mapping data can help elucidate the conduction disease anatomically, attempts to acquire procedural data during past catheter ablations have been unsuccessful to date, and the patient was not interested in further invasive testing.

The patient's ECGs also showed a prolonged PR interval indicating first-degree atrioventricular block. Atrioventricular and interatrial blocks share certain etiologies that are related to the right atrium—up to 41% of patients with atrial flutter have concomitant PR interval prolongation caused by right intra-atrial conduction delay.² Conduction disturbance in atrioventricular block is most often due to idiopathic fibrosis and sclerosis, while other common causes include ischemic heart disease, ablation procedures, and antiarrhythmic medications (eg, metoprolol and amiodarone), all of which were present in this patient.^{3,4} In the literature, evidence is lacking as to whether amiodarone leads to prolongation and wide splitting of P waves in humans. Nonetheless, the reduced amiodarone dosage in this patient did not lead to any ECG change over 3 months.

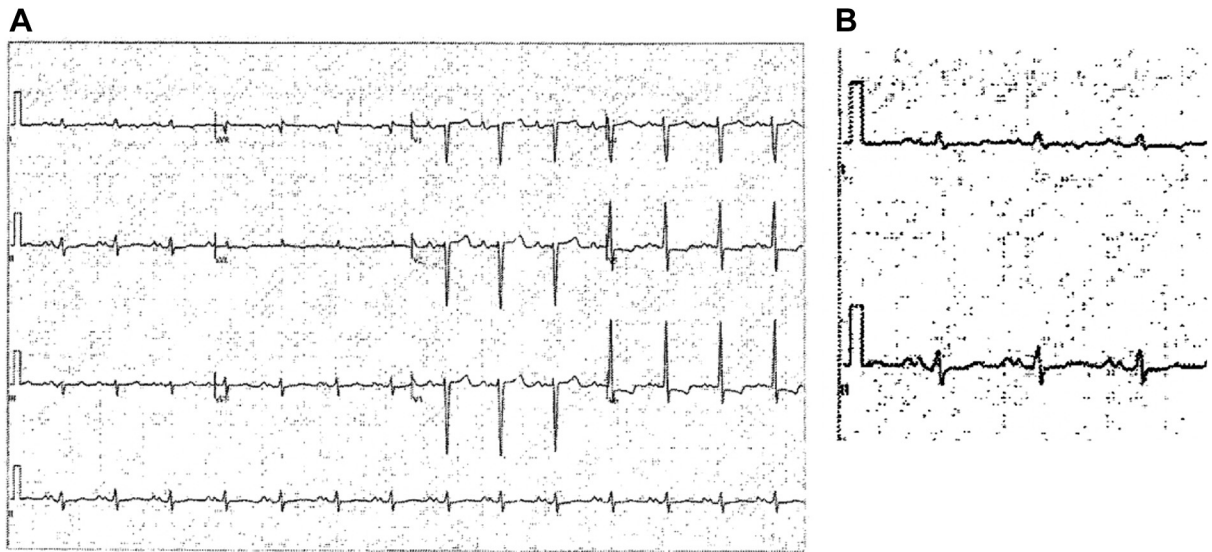


Figure 2 Electrocardiograms prior to the patient's second catheter ablation 5 years ago (A) and in the year after the procedure (B) showing sinus rhythm, P wave splitting, and PR prolongation that were less pronounced compared to current findings.

Cases of prolonged and widely split P waves have been sporadically reported over the past decades. In 1997, double P waves separated by 400 ms were found in a patient with syncope but otherwise no known history of cardiovascular disease. ECG also demonstrated sinus nodal dysfunction, right bundle branch block, and left anterior fascicular block, suggesting pan-conduction system disturbance. An electrophysiology study localized a long line of conduction block in the upper and lower parts of the right atrium.⁵ In a patient with familial *PRKAG2* cardiomyopathy, a hereditary glycogen storage disease, extreme interatrial conduction block leading to split P waves was observed.⁶ In contrast to these cases, we were able to identify in 1 single patient multiple coexisting potential causes of prolonged and markedly split P waves, and leverage longitudinal data points from patient history, ECGs, and imaging studies spanning more than a decade to further evaluate possible etiologies.

Interatrial block may be an under-recognized entity. The need for accurate identification of its ECG patterns, distinguishing between causes, and understanding its clinical implications was described in a consensus report published in 2012.¹ Several statements in this report are pertinent to the present case. Interatrial blocks can progress from lower (partial) to higher degrees (advanced). The characteristic ECG pattern of bimodal P waves in leads I and aVL and biphasic (+/-) P waves in inferior leads, based on deductive ECG-vector cardiography data, are a result of blocked electrical impulse in the upper and middle part of the interatrial septum, the Bachmann bundle zone, and/or the upper left atrium, causing retrograde left atrial activation via myocardial connections near the coronary sinus. A longer P wave duration or higher-degree interatrial block has been associated with risk for supraventricular arrhythmias, eg, atrial fibrillation and flutter, as well as cardiovascular and all-cause mortality.^{7,8} The perioperative P wave morphology and duration

harbor prognostic value in the radiofrequency ablation of atrial fibrillation and flutter.^{9,10} Interatrial conduction delay is associated with prolongation of not only P waves but PR interval. Of note, while interatrial conduction block can often be considered pathognomonic of left atrial enlargement, they do not invariably coexist. In light of all these, prompt recognition of the ECG pattern discussed herein may inform clinical practice.

Conclusion

This case of a patient with widely split P waves in the setting of a complex cardiac history is presented as an educational exercise to understand the etiology and pathophysiology of abnormal P wave morphology. Extremely prolonged and widely split P waves may result from interatrial conduction delay from atrial enlargement or remodeling, changes secondary to radiofrequency ablation, or alteration in conductive tissue properties with medication use. Differentiating between these causes may require thorough history-taking and a comprehensive review of years of ECG and imaging data.

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