



Atrial Fibrillation in a Patient with Interatrial Block after Successful Atrial Flutter Ablation: May Interatrial Block Be a Link for a Continuum of Atrial Arrhythmias?

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Abstract

Background: Electrocardiographic interatrial block (IAB) typically exists when a conduction delay over the Bachmann's bundle is present. By analogy to other types of block, there is a continuum of IAB severity; partial IAB is defined as a P-wave duration 120 ms and advanced interatrial block (aIAB) is defined as a P-wave duration 120 ms in conjunction with biphasic P-wave morphology in inferior leads II, III, and aVF. Moreover, IAB seems to be a key factor in the genesis of atrial fibrillation (AF). On the contrary, it is not clear if the presence of advanced IAB may be associated with an occurrence of AF after cavotricuspid atrial flutter ablation in patients without previous episodes of atrial fibrillation.

So, may be useful to evaluate on surface electrocardiogram the P-wave morphology and duration in sinus rhythm after the ablation procedure just to identify patients at high risk for atrial fibrillation development. Advanced IAB is frequently associated with atrial tachyarrhythmias, and it was found to predict AF and may be helpful for clinical decisions.

Case presentation: In this case involving a man who underwent a typical atrial flutter ablation, we documented that aIAB during the post-ablation period which seems to induce atrial fibrillation episodes. To our knowledge, it is unusual to detect an aIAB after cavotricuspid atrial flutter ablation and subsequently associate it with a new onset of AF.

Conclusions: Our observation supports a possible aIAB induction of AF after cavotricuspid atrial flutter ablation and potentially may representing a link in the continuum of atrial arrhythmias. P-wave morphology and duration should be better evaluated soon after AF ablation to better stratify patients who are at high risk for atrial fibrillation development and eventually prolonged anti-coagulation therapy.

Keywords: *interatrial block, atrial flutter, atrial fibrillation, cavo-tricuspid isthmus ablation*

Introduction

Catheter ablation of the cavo-tricuspid isthmus (CTI) is an established, curative first-line therapy for patients with typical atrial flutter (AFlu), with success rates exceeding 90%. Atrial fibrillation (AF) after successful typical flutter ablation varies from 8-38% ^[1,2] and is associated with previous AF, structural heart disease with reduced ejection fraction and use of a higher number of drugs before ablation and the follow up duration ^[3]. These patients have anisotropic conduction and intra-atrial conduction blocks that predispose them to initiation and maintenance of AF ^[4].

The above appears to reflect a common underlying substrate for both types of arrhythmia ^[5]. On the other hand an association between aIAB, supraventricular arrhythmias, and poor left atrium contractility has been reported. The diagnosis of aIAB is easy to perform using the surface electrocardiogram (ECG). IAB is partial when the P-wave duration is ≥ 120 ms, and advanced if furthermore the P-wave presents a biphasic pattern in the inferior leads i.e. II, III and aVF. The advanced pattern, where there is a total block of Bachmann's region and the conduction towards the left atrium, occurs from the lower part of the right atrium with a caudo-cranial retrograde direction mainly through the coronary sinus, and to a

lesser extent through the fossa ovalis [6,7]. In the present case an aIAB was observed six months after a successful AF ablation procedure with subsequent new onset AF episodes.

Case Presentation

A 67-year-old man with no prior significant past medical history presented at the medical office for some discontinued palpitations not better defined. His electrocardiogram (ECG) revealed normal

sinus rhythm at 70 bpm with occasional premature ventricular complexes. His physical exam showed a minor diastolic heart murmur 1/6 and normal pressure values around 130/85 mmHg. Consequently, he was referred to the hospital for echocardiographic evaluation and cardiologic consultation. Some days later a new episode of palpitations, dizziness and a pre-lipothimic event led him to the emergency room. This time, physical examination revealed an irregular pulse and his ECG showed AFLu (Figure 1).

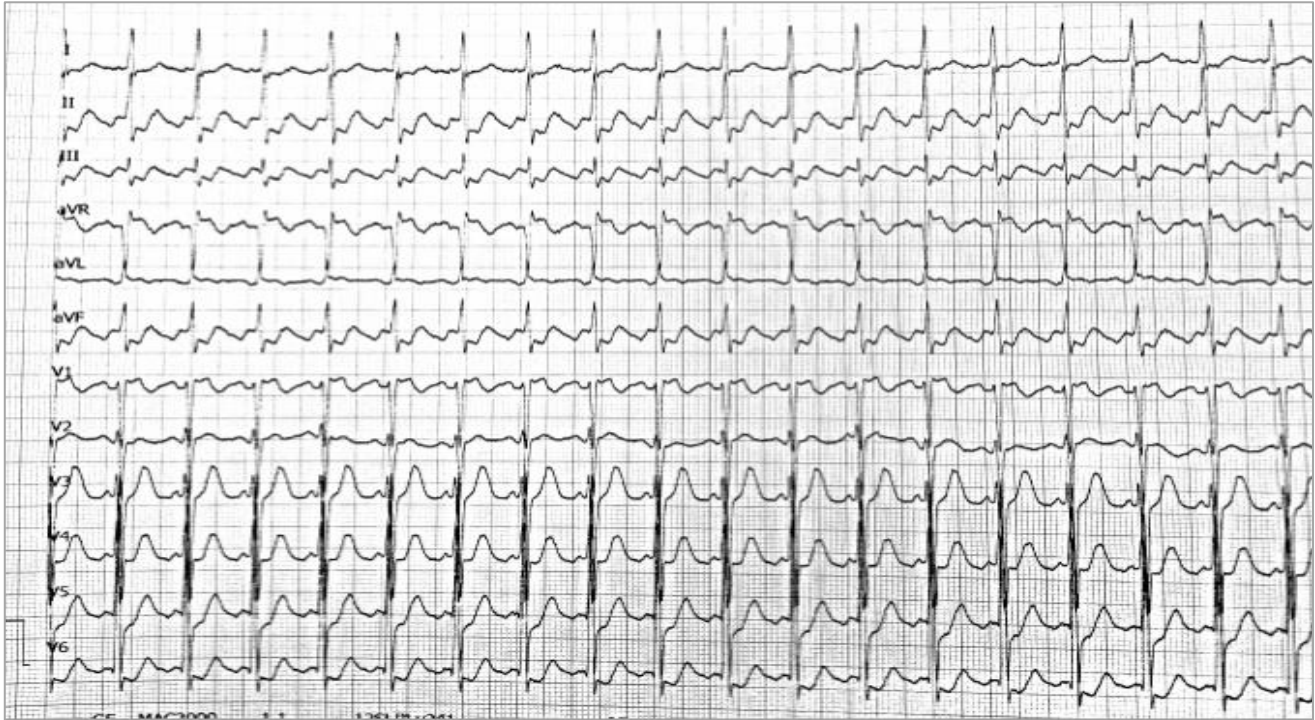


Figure 1: The ECG showing atrial flutter 131 bpm.

Cardiologic evaluation demonstrated a normal imaging study with normal atrial dimensions: 3.7 cm. Also, a normal right atrial size, intact atrial septum, normal biventricular systolic function, and a small aortic regurgitation was observed. Direct oral anticoagulation (DOAC) and antiarrhythmic therapy with propafenone 150 mg three times daily was commenced and the patient was doing well. Twenty days later a relapse of AFLu was observed and ablation therapy was proposed, which was well accepted by the patient. Two days later he successfully underwent a CTI ablation. Based on our protocol, the patient was prospectively followed for three months while under DOAC therapy. Sinus bradycardia (54 bpm) was observed in a follow-up ECG, and a careful observation of the

P-wave morphology, duration, and voltage was obtained revealing an aIAB alteration with a P-wave duration of 135 ms, and the polarity was bimodal in the inferior leads (Figure 2). By applying a previously described electrocardiographic morphology-voltage-P wave (MVP) ECG risk score [9] for the prediction of AF we obtained a score equal to 5 (high probability risk of AF). The above score is a simple electrocardiographic evaluation of P-wave and prediction of AF. In fact, a subsequent Holter ECG monitoring evaluation confirmed the presence of short episodes of AF with spontaneous interruption (Figure 3). Based on these last findings the patient remained on DOAC therapy. It is of note that the CHA2DS2VASc score was zero.



Figure 2: Sinus rhythm after episodes of AF in the period after flutter ablation.

Note the interatrial conduction delay of 135 ms (aIAB) and the biphasic morphology of P-waves in the inferior leads.

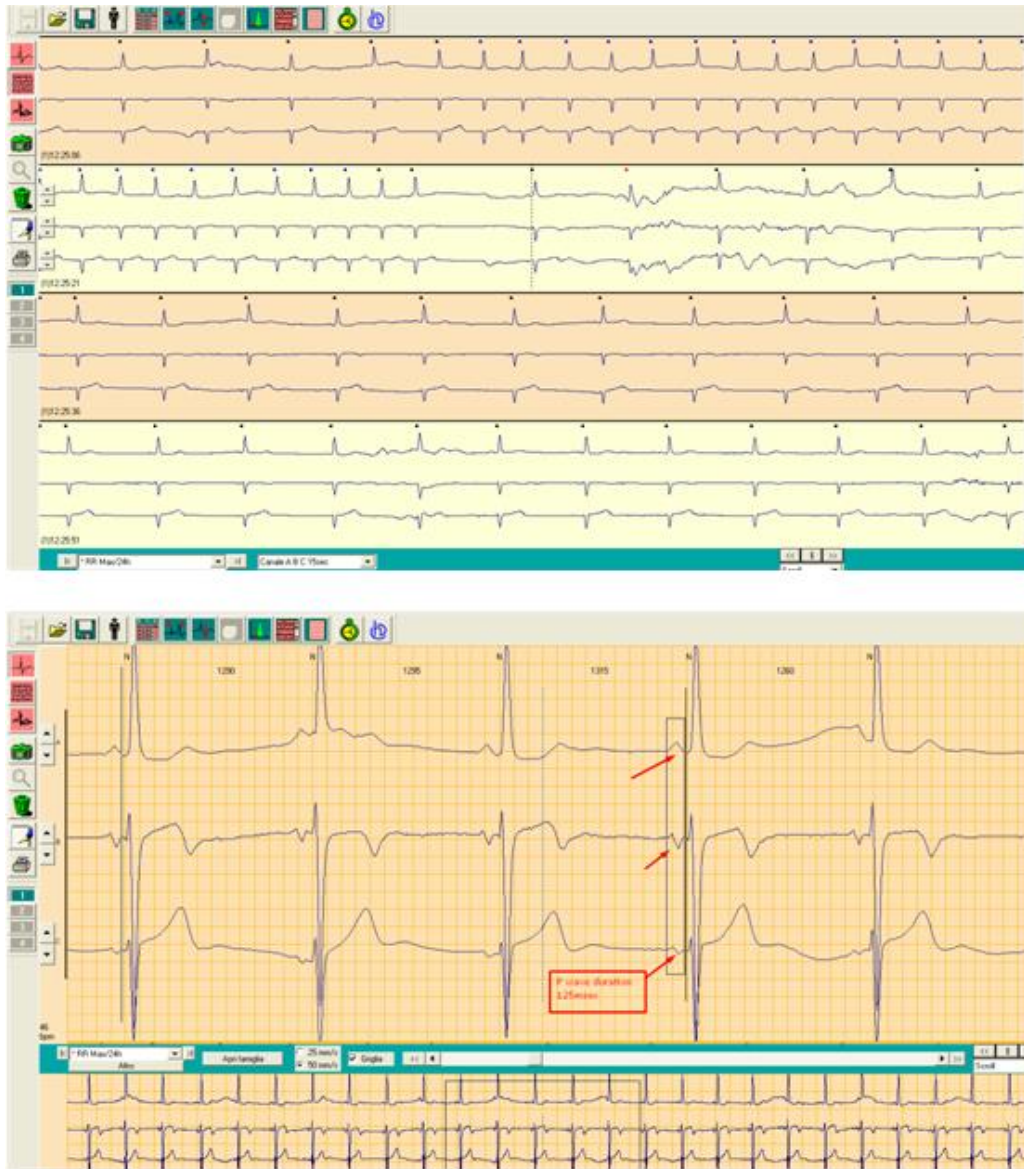


Figure 3: AF episodes in a Holter ECG monitoring in the period after successful AFlu ablation. P-wave duration during sinus rhythm in a Holter ECG monitoring was 125 ms. Here the biphasic morphology of the P-wave is more apparent. Differences in P-wave duration are due to the different days of observation, the semiautomatic measurement and the dynamic registration itself.

Six months later, a 24-hour Holter ECG monitoring confirmed the presence of short episodes of AF with spontaneous cessation (**Figure 3**). The patient, however, was asymptomatic and his physical examination was unremarkable. Subsequently, the beta blocker bisoprolol at 2.5 mg daily was added into the treatment plan. Moreover, a transthoracic echocardiogram was negative for thrombus or atrial dilation and the interatrial septum was intact. The systolic biventricular function was normal. Meanwhile, the patient was allowed to resume his normal daily activities. Some days later the patient complained of palpitations after a short period of cycling. A watch monitor worn by the patient reported an atrial fibrillation episode of 4 hours in duration. The patient was moved to the emergency room where an immediate successful electric cardioversion was performed. Following this we decided to prolong DOAC therapy for six more months.

Discussion

Catheter ablation of the CTI is successful in the long-term for approximately 91% of patients with AFlu if bidirectional CTI block was achieved, and 76% if bidirectional block was not achieved [8,9]. The reported incidence rate of AF after AFlu ablation

is not well known. From previous studies, AF incidence rate depends on the previous presence or not of AF history and the follow up duration. At the same time a structural heart disease and larger left atrial diameter were more common in the patients who developed AF [10].

On the other hand, prolonged (>120 ms) intra-atrial conduction time predicted new-onset AF after ablation in this group. Similarly, in another study, aIAB was a key predictor for high risk of new-onset AF after a successful CTI ablation in patients with typical AFlu [11]. Therefore, the optimal duration of anticoagulation therapy is unknown, and also it has been proposed that isolation of the pulmonary veins should be considered at the time of cavo-tricuspid isthmus ablation. Consequently, the identification of these patients with a substrate for AF development, like aIAB, is helpful for clinical decisions. The appearance of aIAB is frequently associated with atrial tachyarrhythmias, and it was found to predict AF in many different clinical scenarios [12]. Delayed conduction between the right and left atria (LA) induces interatrial dyssynchrony, which leads to electrical heterogeneity in the LA. This condition is known to contribute to AF manifestation and maintenance. Accordingly, interatrial dyssynchrony may induce a prolonged interatrial

conduction time in concomitant LA growth and elongation of the fibers composing the Bachmann's region. On the other hand, an increase in the prevalence of IAB [13] is probably due to the increase in the degree of fibrosis and fatty infiltration that occurs with aging and this can impair the conduction at the auricular level [14,15]. It is well known that an increase in the size of the extracellular matrix, at the level of the atrial interstice, would favour the slowing of the impulse and with this the appearance of different degrees of IAB. Additionally, atrial fibrosis generates an increase in the rigidity of the cardiac wall, resulting in a deterioration of cardiac function and, consequently, affecting the conduction of the nerve impulse [16], thus increasing the risk of IAB development. Accordingly, in a previous study, the presence of aIAB was associated with a higher risk of AF after CTI ablation in patients with typical AFlu and no prior history of AF. Over a mean follow-up time of 30.5+/-15.3 months (median 30 months), 57 patients developed new-onset AF (46.7%). The incidence of AF was greater in patients with aIAB (71.4%) compared to those without aIAB (39.4%, p=0.003). After multivariate analysis, aIAB remained statistically significant (OR 2.9, 95% CI 1.02-8.6; p<0.04) [17].

Likewise, in another study, the presence of aIAB was a strong predictor of new-onset AF post-cavo-tricuspid flutter ablation, and the association remained significant in a multivariate analysis. This study showed that a prolonged P-wave duration alone was insufficient to predict AF. This is because a P-wave >120 ms may also be due to a delay in the right atrium. Furthermore, the inhomogeneous LA activation, evidenced by the biphasic P-wave, is probably more important for the prediction of AF genesis than the total atrial activation time [18]. Consequently, the utility of the P-wave morphology and duration in sinus rhythm was realized, since they can be readily analyzed in patients after the ablation procedure, providing indirect information not only about atrial size but also, and more importantly, about atrial remodeling [19]. The pathophysiologic mechanism was first described by Bayes de Luna in the early 1980s who postulated that P-wave biphasic morphology in the inferior leads reflected predominant caudo-cranial activation of the LA through the coronary sinus [20]. This conduction way has been later confirmed using endocardial mapping [21].

On the other hand, regarding the pathophysiologic connection of CTI-dependent AFlu and AF another hypothesis has been described. CTI-dependent AFlu starts as AF that organizes into a macro-reentrant circuit, due to the development of functionally blocked lines between the two venae cavae. In fact, in these patients, as suggested by Waldo and Feld [7], AF has always been present, and it simply becomes manifest after CTI ablation, because it can no longer evolve into an AFlu. This group of patients may be expected to exhibit extensive atrial remodeling, reflected by an increased P-wave duration, enlarged LA dimensions, tissue fibrosis, and impaired atrial mechanical function.

From the above mentioned mechanisms we can argue that heterogeneous electrical activation of the LA results in impaired LA mechanical function, interatrial dyssynchrony, and increased susceptibility to AF.

Conclusions

In conclusion, the presence of aIAB after typical flutter ablation may induce new onset of atrial fibrillation. Then, in this pathological frame P wave morphology and duration like aIAB may potentially representing a link in the continuum of atrial arrhythmias. As a result the last condition should be better

evaluated soon after AFlu ablation to better stratify patients who are at high risk for atrial fibrillation development and potentially for prolonged anticoagulation therapy.

Ethics approval and consent to participate

No ethical committee approval was required for this case report by the Departments, because this article does not contain any studies with human participants or animals.

Informed consent

Consent of EKG exam was obtained from the patient included in this study.

Consent for publication

Not required as each EKG and event recorder image is anonymized and therefore the confidentiality or personal data is guaranteed.

Conflicts of interest

The authors report no conflicts of interest or funding support.

Authors' contributions

AA has drafted the work and revised the final presentation, DD has treated the patient and followed him and provided follow up data, and GA followed the patient and revised the last draft.

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