

A Case Report of Rate Control With Ivabradine in a Patient With Refractory Atrial Tachycardia and Heart Failure

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Case report

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Abstract

Background: Already known ivabradine's effects in decreasing atrial spontaneous activity and in pulmonary veins, the focal atrial tachycardia, New information is that it is candidate for re-entrant atrial arrhythmias.

Case presentation :An 85-year-old woman with a history of underlying ischemic cardiomyopathy complained of worsening heart failure symptoms due to rapid atrial tachycardia, which was resistant to several rate control drugs but responded well to ivabradine. An electrophysiology study demonstrated a roof dependent macro-reentrant tachycardia of the left atrium. Linear ablation at the left atrium roof resulted in termination of the tachycardia.

Conclusion: Ivabradine can be used as an effective rate control drug of reentrant atrial tachycardia.

Background

Atrial tachycardia is common in heart failure with reduced ejection fraction (HFrEF). It can cause uncontrolled heart failure and is often difficult to treat. Cardioversion therapy is preferred but thrombus in the left atrial appendage is common. Rate control became reasonable choice in extremely symptomatic patients having a rapid ventricular rate. Unfortunately, Due to hypotension and other side effects, there are limited options. We try to provide better options for achieving ventricular rate control.

Case Presentation

An 85-year-old woman with ischemic cardiomyopathy, hypertension, and diabetes mellitus was referred due to her recurrent heart failure accompanied by rapid atrial arrhythmia. Pulmonary vein isolation with radiofrequency ablation was performed successfully in February 2019. The patient was prescribed betaloc ZOK 23.75mg once daily. She had no other antiarrhythmic medications because a slow resting heart rate of ≤ 60 bpm was present. She remained asymptomatic and in sinus rhythm for a year. Echocardiography revealed the left atrial diameter was 44 mm, the left ventricle end diastolic diameter was 54 mm, and her LVEF was at 40% during regular follow up testing.

In April 2020 the patient was admitted several times for heart failure. Each admission was accompanied by rapid atrial arrhythmia. ECG showed atrial tachycardia (AT) with a 250 bpm atrial rate and 2:1 AV conduction (Fig. 1). The left ventricle enlarged to 63mm while LVEF decreased to 22%. Atrial tachycardia is closely related to heart failure deterioration and electrical cardioversion was attempted.

Transesophageal echocardiography (TEE) showed left atrial appendage thrombosis. Betaloc ZOK 23.75mg once daily, diltiazem 15mg thrice daily, digoxin 0.125 mg tertian, and even intravenous amiodarone were given to control ventricular rates. Her average heart rate remained 120bpm meanwhile hypotension appeared. Amiodarone was discontinued due to it being less effective and the presence of a severe hepatic injury.

The guidelines recommendations on antiarrhythmic drugs were followed, but a number of dilemmas remained. The patient consented to ivabradine treatment with was 5mg twice daily was off-label. Four [4] days after beginning this treatment, ventricular rate reduction and other symptom relief was obtained (Fig. 2). A dynamic electrocardiogram showed average heart rate of 62bpm throughout the day. The heart failure was immediately relieved and the patient was discharged .

One month later, in May 2020, an electrophysiology study [EPS] was performed after an atrial appendage thrombus resolution was confirmed by TEE. Ensite Navx 3D mapping showed reentry isthmus on the left roof. Reentry circumference in left atrial was 260ms. Tachycardia ceased after ablation of the left roof (Fig. 3). Ivabradine was post-op discontinued. The patient is now [January 2021] at 8-months post-ablation and continues to be symptom free.

Discussion And Conclusions

For this patient, electrocardioversion was unquestionably the best option, but thrombus present in the left atrial appendage made that impossible. Here the international guidelines for the treatment of supraventricular tachycardia ¹ by using recommended antiarrhythmic drugs such as digoxin, beta-blockers, and calcium channel blockers was performed did not obtain arrhythmia cessation or significant rate reduction. Ivabradine played an important role by slowing ventricular rate of reentrant atrial tachycardia.

Ivabradine's effects in decreasing atrial spontaneous activity and in pulmonary veins, the focal atrial tachycardia, has been reported^{2,3}. The latest Guidelines recommend considering ivabradine if the other measures fail in the treatment of focal atrial tachycardia ¹. The suspected mechanism is that I(f) is carried by hyperpolarization-activated, cyclic nucleotide-gated (HCN) channels. HCN4 is the predominant subtype present in the sinoatrial, and atrioventricular(AV) nodes^{4,5}. Ivabradine can slow tachycardia by affecting HCN4 channels present in the AV node. This is what happened in atrial fibrillation in the experimental model⁶. It can be supposed that controlling ventricular rate may occur in arrhythmia generated by a reentry mechanism.

In the instant case, ventricular rate decreased by day four of ivabradine treatment. P-wave morphology similar to that of the tachycardia on ECG suggested that atrioventricular conduction had decreased due to ivabradine. EPS confirmed left atrial reentry tachycardia. The circumference of the reentrant circuits changed little and indicates that the action is on the atrioventricular node rather than the atrial.

Ivabradine is a selective I(f) inhibitor currently used to manage patients with stable angina pectoris⁷ or heart failure⁸. It acts in a dose dependent way and slows the ventricular rate with no effect on myocardial contractility. This is important for treating heart failure, especially for those with reduced LVEF. Atrial macro-reentry related tachycardias are common in the elderly or in patients with structural heart disease. Ivabradine is a candidate medication when atrial reentry tachycardia resistance to routine drugs and cardioversion is not possible.

Abbreviations

AT: atrial tachycardia

AV: atrioventricular

CLBBB: Complete left bundle branch block

ECG: electrocardiogram

EPS: electrophysiology study

HCN: hyperpolarization-activated, cyclic nucleotide-gated

HFrEF: heart failure with reduced ejection fraction

LVEF: left ventricular ejection fraction

TEE: Transesophageal echocardiography

Declarations

Ethics approval and consent to participate

The case report was approved and supervised by the ethics committee of Beijing Tsinghua Changgung Hospital.

Consent for publication

Written informed consent for publication of patients' clinical details and clinical images was obtained.

Competing interests

The authors declare that they have no competing interests.

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Contributions

Lanting Zhao and Rong He contributed equally to this paper. All authors read and approved the final manuscript.

Availability of data and materials

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

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Not applicable

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Figures

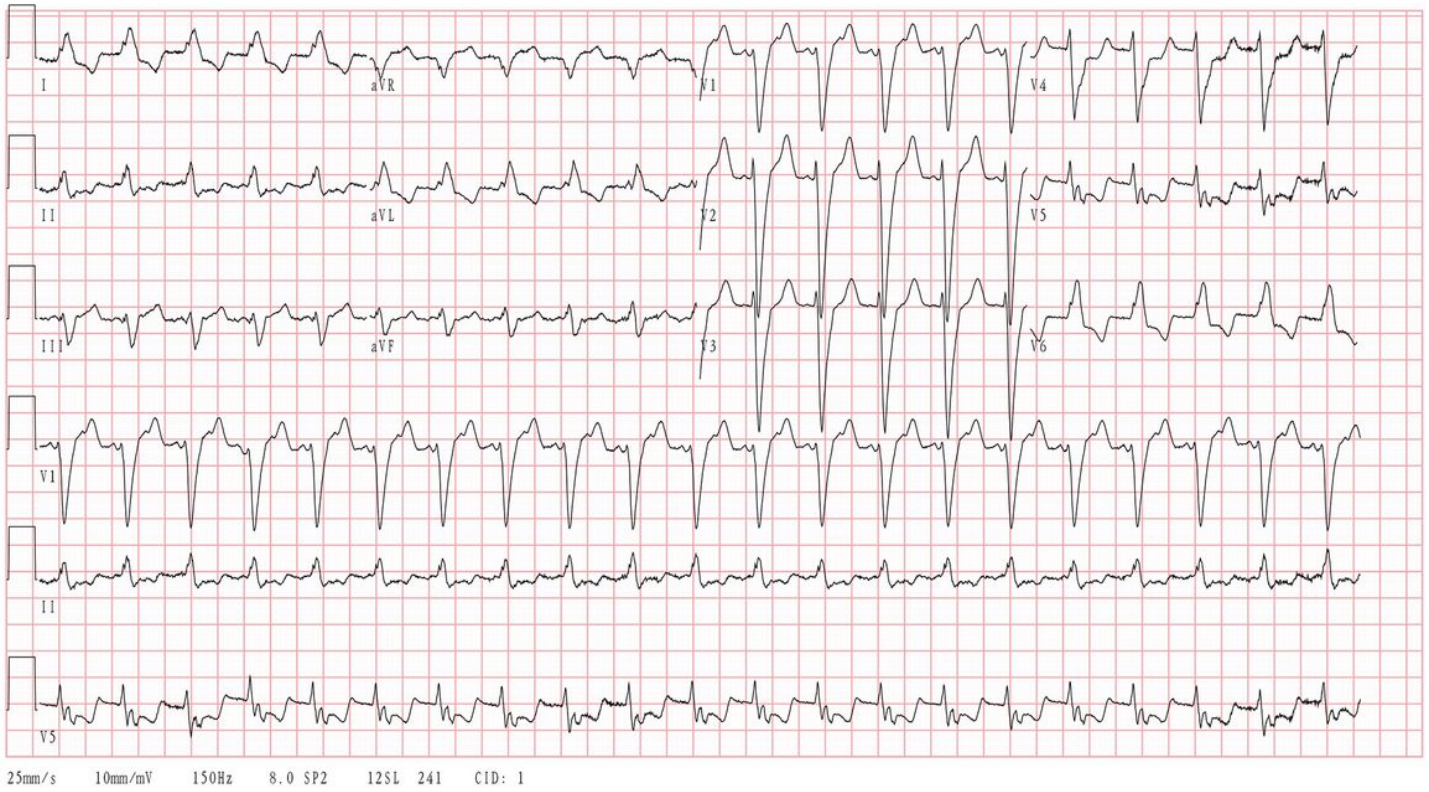


Figure 1

ECG showing heart failure: atrial tachycardia, atrial rate 250 bpm, 2:1 AV conduction; CLBBB

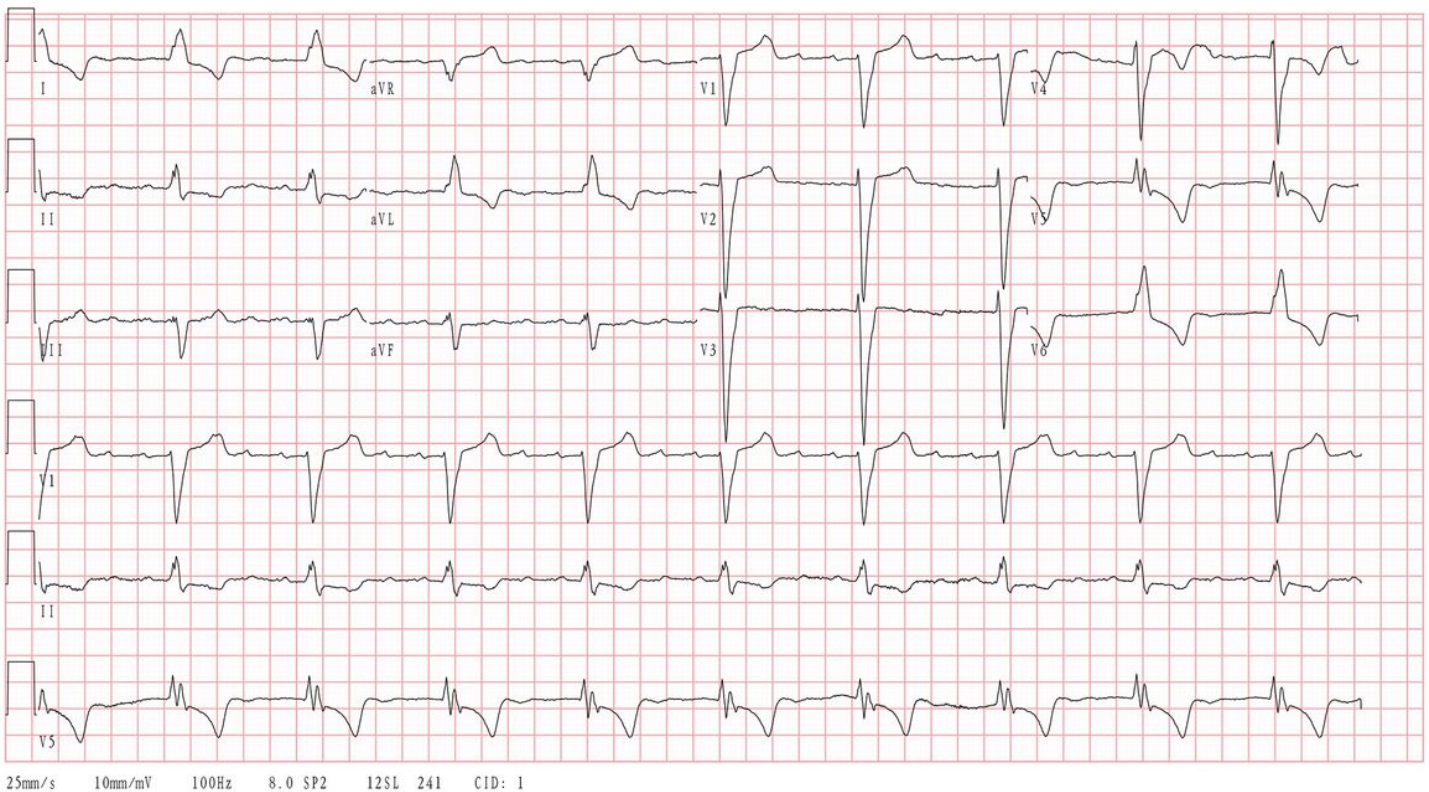


Figure 2

ECG on fourth day of starting ivabradine 5mg twice daily: atrial tachycardia, atrial rate 230bpm, 4:1 conduction, CLBBB



Figure 3

EPS

Supplementary Files

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