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

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Body

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: 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 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 (Figure 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 (Figure 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 . Ivabradine was post-op discontinued. The patient is now [January 2021] at 8-months post-ablation and continues to be symptom free.

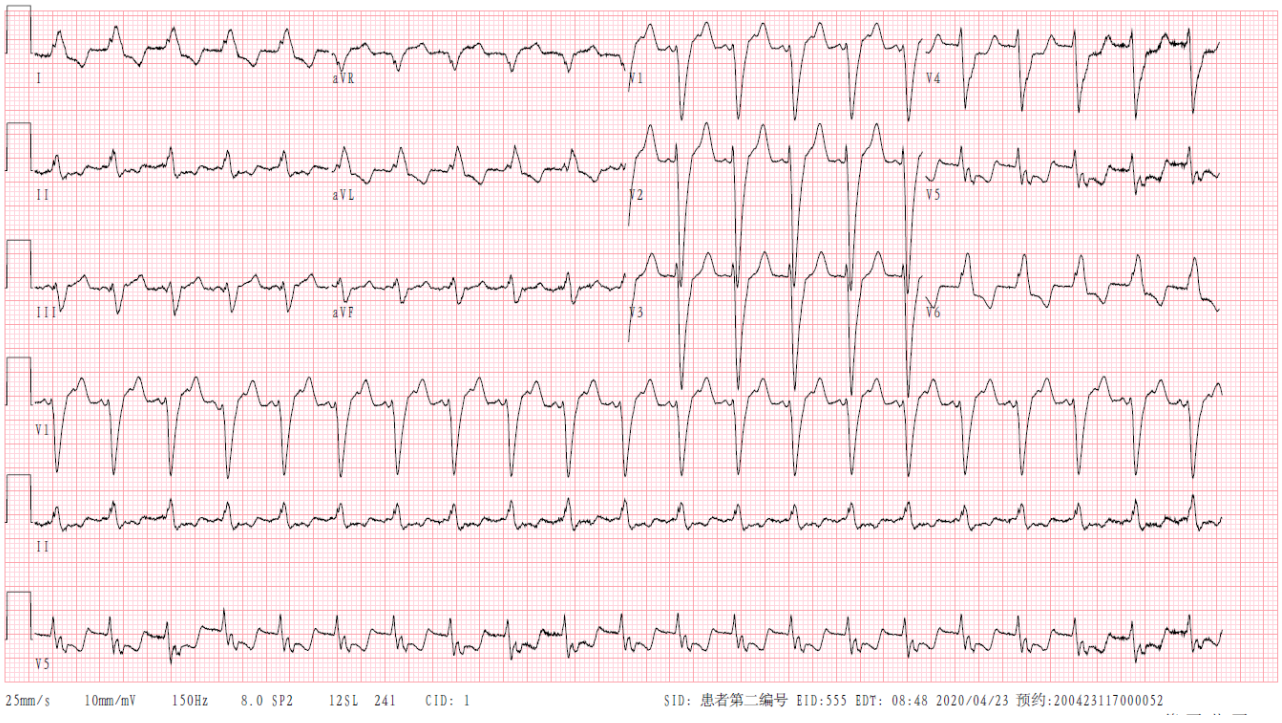
Discussion and Conclusion: For this patient, cardioversion was unquestionably the best option, but thrombus present in the left atrial appendage made that impossible. Here the international guidelines for the treatment of supraventricular tachycardia [1] by using recommended antiarrhythmic drugs such

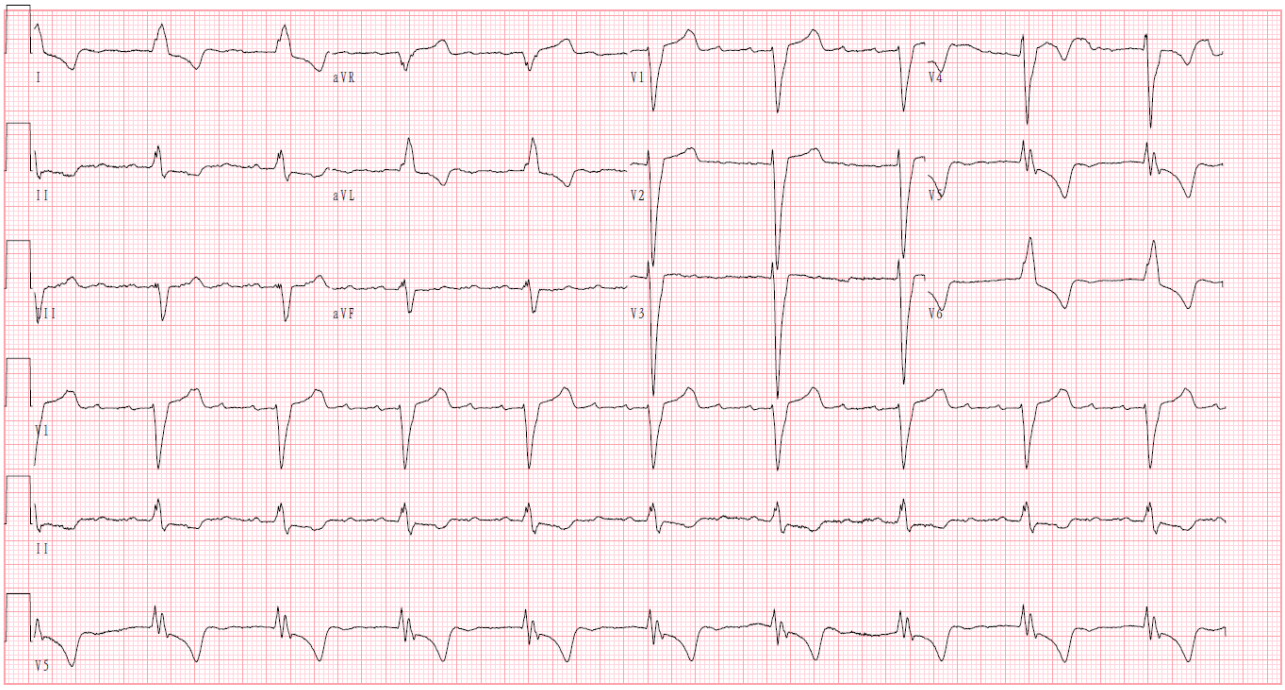
as digoxin, beta-blockers, and calcium channel blockers was performed did not obtain arrhythmia cessation or significant rate reduction. Ivabradine played an important role by slowing ventricular rate of reentrant atrial tachycardia.

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 model[6]. 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 pectoris[7] or heart failure[8]. 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.





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