CASE REPORT

Wide Complex Tachycardia in the Presence of Class I Antiarrhythmic Agents: A Diagnostic Challenge

Bhaskar Bhardwaj, M.D., Ralph Lazzara, M.D., and Stavros Stavrakis, M.D. Ph.D.

From the Department of Medicine, Cardiovascular Section, University of Oklahoma Health Sciences Center, Oklahoma City, OK

We present two patients with paroxysmal atrial fibrillation on class 1C antiarrhythmic drugs without concomitant atrioventricular (AV) nodal blocking agents who developed atrial flutter with 1:1 AV conduction. Their electrocardiogram revealed wide complex tachycardia with rates >200/minute. Atrial flutter with 1:1 conduction in the presence of class IC antiarrhythmic drugs may present a diagnostic challenge. These cases illustrate the importance of coadministering an AV nodal blocking agent with class IC antiarrhythmic agents in patients with atrial fibrillation. The differential diagnosis of wide complex tachycardia in patients taking class IC agents should include atrial flutter with 1:1 AV conduction.

Ann Noninvasive Electrocardiol 2014;19(3):289–292

wide complex tachycardia; antiarrhythmic agents; atrial flutter

Atrial flutter with 1:1 atrioventricular (AV) conduction is a rare occurrence, but may be seen with the use of class 1 antiarrhythmic drugs, such as flecainide and propafenone.^{1–4} It is often misdiagnosed as ventricular tachycardia because of wide QRS complexes seen in electrocardiogram (ECG). Ventricular tachycardia has also been described with the use of these drugs.⁵ Differentiating between supraventricular tachycardia with aberrancy and ventricular tachycardia represents a diagnostic challenge, but has significant clinical implications with regards to treatment. We report two cases of atrial flutter with 1:1 conduction, which presented as wide complex tachycardia in the presence of class IC antiarrhythmic drugs.

CASE 1

A 72-year-old male with history of paroxysmal atrial fibrillation and no structural heart disease, who was recently started on propafenone, presented to the emergency room with complaints of palpitations associated with dizziness. His vital signs included blood pressure of 80/60 mm Hg

and heart rate of 202 beats/minute. A 12-lead ECG showed wide complex tachycardia with a rate 207 beats/minute, which was initially interpreted as ventricular tachycardia (Fig. 1A). Because the patient was found to be hyperkalemic (K = 5.5 meq/L), he was given intravenous calcium gluconate, which caused a change in the morphology of the tachycardia to a narrow QRS complex (Fig. 1B). The second ECG, which was obtained 15 minutes after the first one, clearly showed atrial flutter with 2:1 AV conduction, although it should be pointed out that the flutter waves were somewhat atypical, reflecting the propafenone-induced slowed myocardial conduction. The rate of the atrial flutter waves was the same as the rate of the wide complex tachycardia. Retrospectively, the first ECG was interpreted as atrial flutter with 1:1 AV conduction. Propafenone was stopped, and the patient was given intravenous fluids and intravenous metoprolol, which improved the heart rate to 90 beats/minute and blood pressure to 100/60 mm Hg with resolution of patient's symptoms. Overnight the patient remained in atrial flutter with variable AV conduction. A repeat 12-lead

Address for correspondence: Stavros Stavrakis, M.D., Ph.D., University of Oklahoma Health Sciences Center, 1200 Everett Dr., 6E103, Oklahoma City, OK 73104. Fax: 405-271-7455; E-mail: stavros-stavrakis@ouhsc.edu

Conflict of interest: none.

Figure 1. Electrocardiogram during initial presentation showing wide complex tachycardia (A), which converted spontaneously to atrial flutter with 2:1 conduction. (B) Note that the atrial flutter rate and the rate of the wide complex tachycardia is identical. (C) Electrocardiogram taken 18 hours after the first one, showing atrial flutter with 3:1 conduction. Note that the atrial flutter rate has accelerated, likely because of partial wash out of propafenone.

Figure 2. (A) rhythm strip of the initial presentation showing very broad QRS complexes. (B) Electrocardiogram after cardioversion showed normal QRS complex.

ECG the following morning showed atrial flutter with 3:1 AV conduction (Fig. 1C), now with a faster atrial rate than the previous two ECGs, likely because the effect of propafenone started to dissipate. He was discharged home on oral metoprolol 50 mg twice daily for rate control of atrial flutter and continued oral anticoagulation.

CASE 2

A 54-year-old male with paroxysmal atrial fibrillation was started on metoprolol and flecainide. The patient did not have structural heart disease based on echocardiography. Metoprolol was discontinued later because of sinus bradycardia. Four weeks after discontinuation of metoprolol, the patient presented with palpitations and presyncope. ECG strip showed wide-complex tachycardia at a rate of 230 beats/minute (Fig. 2A). Electrical cardioversion converted the wide complex tachycardia back to sinus rhythm (Fig. 2B). The QRS interval after cardioversion was not prolonged (106 ms). Coronary angiography revealed normal coronaries. After flecainide was discontinued for 3 days, the patient underwent an electrophysiological study, in which no ventricular tachyarrhythmia was induced with programmed

electrical stimulation. However, sustained counterclockwise atrial flutter was easily induced. The wide-complex tachycardia was therefore thought to be caused by administration of flecainide in the absence of an AV nodal blocking agent. In this situation, the atrial flutter was slowed down by flecainide. The patient underwent successful ablation of counterclockwise atrial flutter a week later and is currently asymptomatic.

DISCUSSION

The class 1C drugs flecainide and propafenone are recommended as first line therapy for maintenance of sinus rhythm in patients with recurrent paroxysmal or persistent atrial fibrillation without structural heart disease.⁶ Nonetheless, the risk of proarrhythmia with these drugs is well recognized.⁶ Propafenone is a sodium channel blocker with weak beta adrenoreceptor and calcium channel blocking activity.⁷ Flecainide is also a sodium channel blocker, thus prolonging conduction, with minimal effect on repolarization.⁸ Both drugs depress the cardiac excitability and decrease the conduction throughout the cardiac tissue. The decrease in the atrial flutter rate

to the range of 200 beats/minute can lead to 1:1 ventricular response in the absence of AV nodal blocking agents, such as beta blockers and nondihydropyridine calcium channel blockers.

The differential diagnosis of wide QRS complex tachycardia includes ventricular tachycardia, supraventricular tachycardia with aberrancy and preexcitation.⁹ Many criteria have been proposed to correctly diagnose wide QRS complex tachycardias; however, despite all available criteria, misdiagnosis is still difficult to avoid.⁹ Atrial flutter with 1:1 conduction in the presence of class IC antiarrhythmic agents may present a confusing picture on the ECG with wide QRS complexes. QRS widening is because of 1. Rate-dependent ventricular conduction slowing and 2. Rate-related aberrant conduction in the His-Purkinje system. Rate-dependent ventricular conduction slowing by class IC antiarrhythmic agents is because of use-dependent sodium channel blockade.¹⁰ Drug binding to the sodium channel is facilitated by depolarization, which results in channel opening and inactivation, whereas drug dissociation is facilitated by repolarization, which returns sodium channels to their resting state. Drug effects are more prominent at faster rates because of an increase in the amount of time the sodium channels are in the activated and inactivated states compared to the resting state.¹⁰ Rate-dependent sodium channel blockade may be responsible for the proarrhythmic properties of these drugs.¹⁰ In addition, class IC antiarrhythmic agents can cause rate-related aberrant conduction of a supraventricular tachycardia, including atrial flutter, because of a slowing of conduction through the His-Purkinje system that is exaggerated at faster heart rates (use-dependency).¹¹ Aberrancy usually takes the form of left or right bundle branch block. However, in the presence of IC agents, myocardial conduction is depressed, especially during faster rates (use-dependency), giving rise to a bizarre-looking wide-complex tachycardia, mimicking ventricular tachycardia. In other words, this use-dependency may explain the very broad, bizarre-looking tachycardia our patients presented with, and the normal-looking QRS complex during sinus rhythm. Therefore, the possibility of atrial flutter with 1:1 AV conduction and aberrancy should always be in the differential diagnosis in patients who present with a wide complex tachycardia in the presence of a class IC antiarrhythmic agent, without concomitant AV

nodal blocking agent. It is equally important to administer an AV nodal blocking agent whenever prescribing class IC agents in patients with atrial fibrillation to prevent rapid ventricular response. The differentiation of atrial flutter with 1:1 conduction from ventricular tachycardia has very important clinical implications with regards to treatment, as illustrated by both our cases.

CONCLUSION

Our cases highlight the diagnostic challenge atrial flutter with 1:1 conduction in the presence of a class IC antiarrhythmic agent may present. These cases also illustrate the importance of coadministering an AV nodal blocking agent with class IC antiarrhythmic agents in patients with atrial flutter or fibrillation, to avoid such untoward events.

REFERENCES

1. Brembilla-Perrot B, Houriez P, Beurrier D, et al. Predictors of atrial flutter with 1:1 conduction in patients treated with class I antiarrhythmic drugs for atrial tachyarrhythmias. *Int J Cardiol.* 2001;80:7-15.
2. Crijns HJ, van Gelder IC, Lie KI. Supraventricular tachycardia mimicking ventricular tachycardia during flecainide treatment. *Am J Cardiol.* 1988;62:1303-1306.
3. el-Harari MB, Adams PC. Atrial flutter with 1:1 atrioventricular conduction caused by propafenone. *Pacing Clin Electrophysiol.* 1998;21:1999-2001.
4. Randazzo DN, Schweitzer P, Stein E, et al. Flecainide induced atrial tachycardia with 1:1 ventricular conduction during exercise testing. *Pacing Clin Electrophysiol.* 1994;17:1509-1514.
5. Falk RH. Flecainide-induced ventricular tachycardia and fibrillation in patients treated for atrial fibrillation. *Ann Intern Med.* 1989;111:107-111.
6. Wann LS, Curtis AB, January CT, et al. 2011 accf/aha/hrs focused update on the management of patients with atrial fibrillation (updating the 2006 guideline): A report of the american college of cardiology foundation/american heart association task force on practice guidelines. *Circulation.* 2011;123:104-123.
7. Bryson HM, Palmer KJ, Langtry HD, et al. Propafenone. A reappraisal of its pharmacology, pharmacokinetics and therapeutic use in cardiac arrhythmias. *Drugs.* 1993;45:85-130.
8. Nappi JM, Anderson JL. Flecainide: A new prototype antiarrhythmic agent. *Pharmacotherapy.* 1985;5:209-221.
9. Alzand BS, Crijns HJ. Diagnostic criteria of broad qrs complex tachycardia: Decades of evolution. *Europace.* 2011;13:465-472.
10. Ranger S, Talajic M, Lemery R, et al. Kinetics of use-dependent ventricular conduction slowing by antiarrhythmic drugs in humans. *Circulation.* 1991;83:1987-1994.
11. Goldberger ZD, Rho RW, Page RL. Approach to the diagnosis and initial management of the stable adult patient with a wide complex tachycardia. *Am J Cardiol.* 2008;101:1456-1466.