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Letter to the Editor

Unstable wide complex tachycardia during propafenone therapy

Sir,

A 70-year-old woman was admitted to our Intensive Care Unit (ICU) with the diagnosis of acute renal failure and respiratory failure. Shortly after temporal haemodialysis, she was successfully weaned from mechanical ventilator. On admission to ICU 3 days, she developed an acute atrial fibrillation episode without hemodynamic compromise. Echocardiography showed normal left ventricular ejection fraction. Amiodarone was given intravenously with resultant transition atrial fibrillation to persistent atrial flutter with 2:1 conduction and a ventricular rate of 134 beats/min. She underwent attempted chemical cardioversion of atrial flutter by oral propafenone 600 mg, with gradual slowing atrial flutter rate and variable atrioventricular conduction thereafter (Fig. 1A). Two hours after starting propafenone, she collapsed with rapid

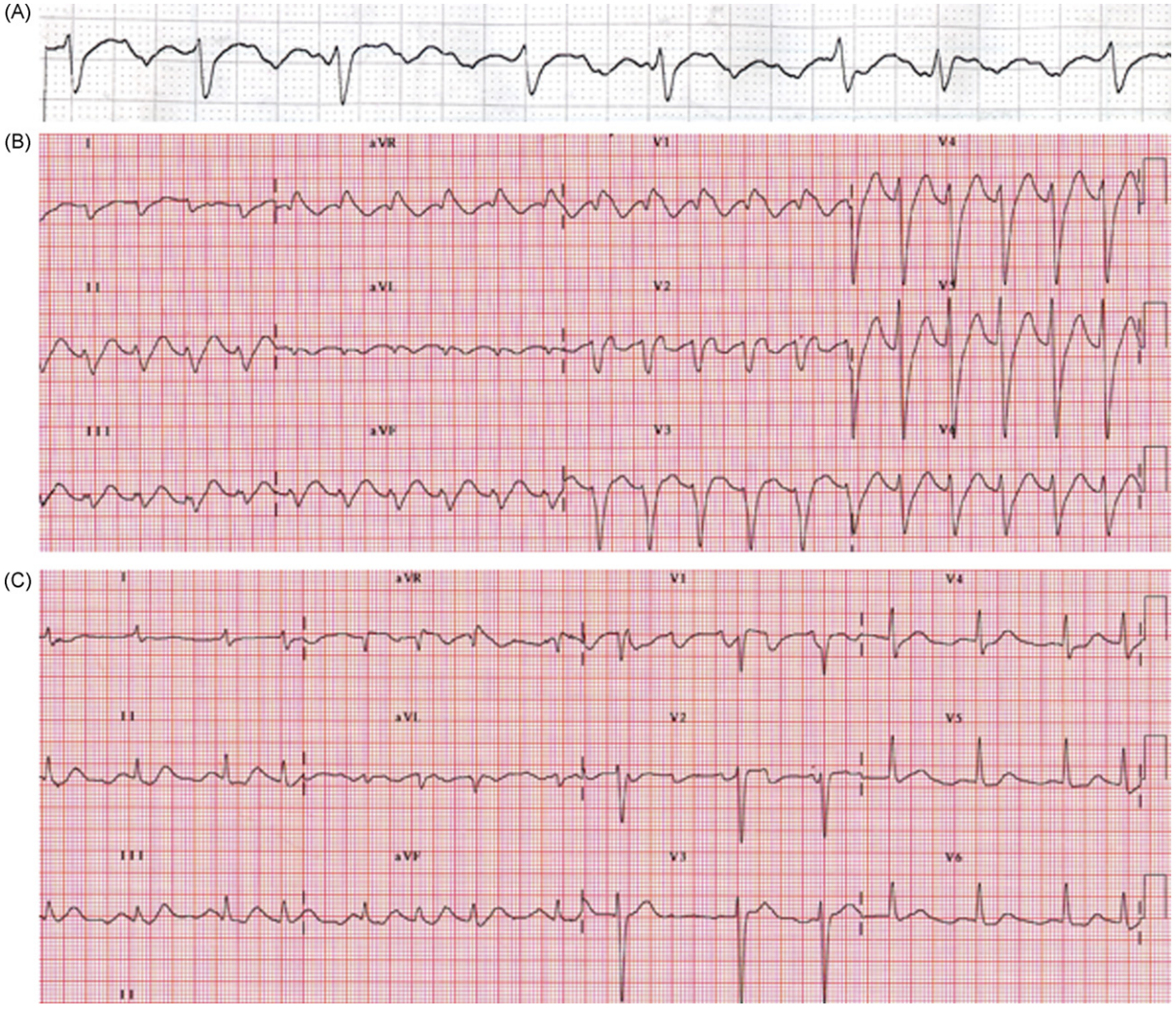

Fig. 1. (A) Electrocardigraphic tracing obtained an hour after propafenone administration showed a atrial rate of 188 beats/min with 2:1 or 3:1 conduction. (B) Wide complex tachycardia observed during propafenone therapy. (C) Identifiable P waves (leads V1–V2) at a rate of 134 beats/min suggested atrial tachycardia with variable atrioventricular conduction. This underscored that wide complex tachycardia was a 1:1 atrial tachycardia with aberrant conduction.

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palpitation and severe hypotension (blood pressure 60/32 mm Hg). Twelve-lead ECG revealed a regular wide QRS tachycardia of rate 134 beats/min (Fig. 1B). On the putative diagnosis of compromised ventricular tachycardia, immediate cardiopulmonary resuscitation with brief chest compression was applied. However, before intending to do DC cardioversion, slowing of the ventricular rate occurred spontaneously during fluid resuscitation, and acute narrowing and near normalization of the QRS complex was observed (Fig. 1C). Thirty minutes later, she converted spontaneously to sinus rhythm accompanied by an increased in blood pressure. The 12-lead ECG showed T wave inversion at leads V1-V3. Coronary angiography revealed left anterior descending artery spasm eliminated by intracoronary administration of isosorbide dinitrate. Thyroid function and electrolytes tests were normal. She was eventually discharged with amiodarone 200 mg twice daily for preventing atrial fibrillation recurrence.

Propafenone, a Class IC antiarrhythmic drug, is a sodium channel-blocking agent with weak β-adrenergic antagonist and calcium antagonist activity. It also exhibits negatively inotropic actions.[1] 1:1 Atrial flutter with accelerated ventricular response during class I antiarrhythmic drugs (e.g. quinidine, flecainide) therapy with reducing atrial rate to the range of 200 beats/min is well recognized.[2–5] The acceleration of the ventricular rate leads to widening QRS complex during tachycardia, representing a diagnostic challenge in differentiating from ventricular tachycardia.[2,4,5]. Wide QRS complex in this case clearly was rate related because it occurred only after a significant increase of the ventricular rate. Moreover, slowing of the ventricular rate was acutely paralleled by narrowing of the QRS complex. This "frequency-depend" effect resulted in a wide complex tachycardia with bizarrely shaped QRS complexes of supraventricular origin, leading to the erroneous diagnosis of ventricular proarrhythmia of propafenone. Propafenone has no vagolytic activity, rather it has β-blocking effects. Despite this, 1:1 atrial tachycardia with extreme aberrant conduction in our case was poorly tolerated. It is therefore imperative that concomitant atrioventricular blocking agents are used both prophylactically or acutely during treatment of atrial tachyarrhythmias with propafenone.

Conflict of interest statement

None declared.

References

1. Zipes DP, Libby P, Bonow RO, et al. Braunwald's heart disease: a textbook of cardiovascular medicine. 7th ed. Philadelphia: Elsevier Saunders; 2005. pp. 725–726.
2. Crijns HJ, Van Gelder IC, Lie KI. Supraventricular tachycardia mimicking ventricular tachycardia during flecainide treatment. Am J Cardiol 1988;62: 1303–6.
3. Ahsan A, Aldridge R, Bowes R. 1:1 Atrioventricular conduction in atrial flutter with digoxin and flecainide. Int J Cardiol 1993;39:88–90.
4. Randazzo DN, Schweitzer P, Stein E, Banas JS, Winters SL. Flecainide induced atrial tachycardia with 1:1 ventricular conduction during exercise testing. PACE 1994;17:1509–14.
5. Wiesfelda ACP, Ansinkb JM, van Veldhuisena DJ, van Gelde IC. Broad complex tachycardia during treatment of atrial fibrillation with a 1c antiarrhythmic drug: Ventricular or supraventricular proarrhythmia? Int J Cardiol 2006;107: 140–1.

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