Lethal Proarrhythmic Effect of Propafenone

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A 67-year-old man with history of paroxysmal atrial fibrillation and known ischemic cardiomyopathy, dyslipidemia, hypertension, and chronic renal failure, was admitted via the emergency room with complaints of palpitations. Past medical history was remarkable for prior myocardial infarction in 2007 followed by percutaneous coronary intervention in the left anterior descending and right coronary arteries. Recent echocardiography showed a left ventricular ejection fraction of 30-35%. During the admission he was found to be in atrial fibrillation with a ventricular rate of 110 bpm (**Fig.** 1) and was given orally a dose of 450 mg of propafenone.

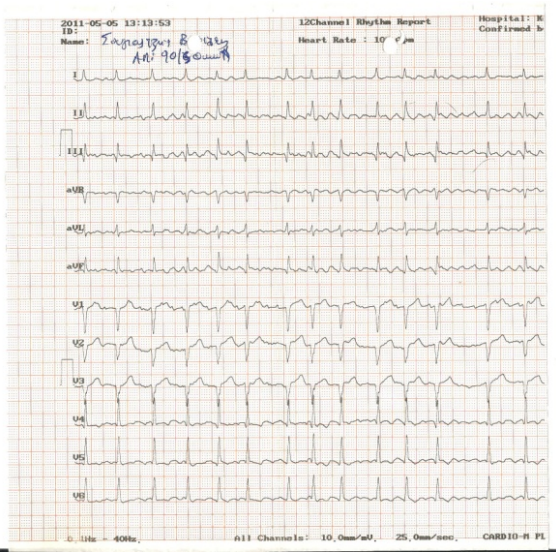

Figure 1. Initial ECG showing atrial fibrillation.

Soon after receiving propafenone, the patient developed dyspnea, precordial heaviness, and hypotension (systolic blood pressure ~70 mmHg). A repeat ECG revealed a wide-QRS tachycardia with a QRS of 200 ms and LBBB-like morphology (**Fig**. 2). Due to hemodynamic instability, the patient was initially electrically cardioverted and IV infusions of lidocaine and amiodarone were started. However, the tachycardia was recurring. Patient was placed on inotropic support with IV dopamine, dobutamine and noradrenaline, while additional DC shocks were required. A temporary pacing wire was introduced and used for overdrive pacing.

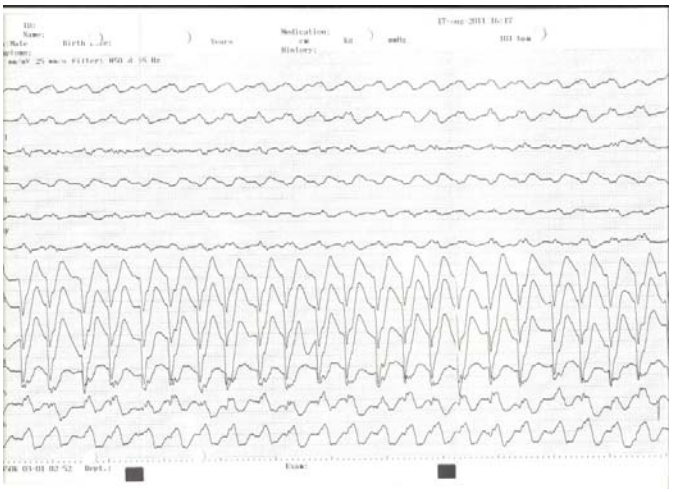

Figure 2. ECG of the wide-QRS tachycardia noted after intake of propafenone demonstrating an LBBB-like QRS morphology.

Despite aggressive therapy and throughout hospitalization the patient remained in incessant ventricular tachycardia (**Fig**. 3) that was also treated with overdrive pacing, but the patient succumbed within 36 hours due to cardiogenic shock and multiple organ failure.

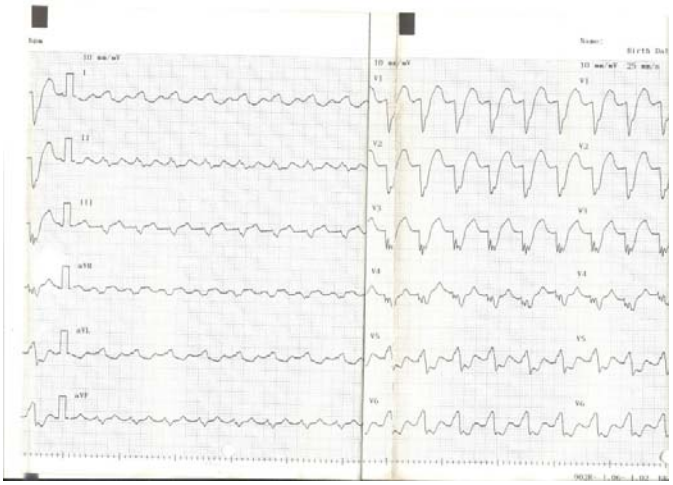

Figure 3. The patient remained in incessant wide QRS tachycardia with LBBB like QRS morphology throughout his hospitalization

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Propafenone prolongs refractoriness and slows sinoatrial node frequency, conduction to the atria and the atrioventricular node. It can aggravate His-Purkinje block and also has negative inotropic effect due to its beta-adrenergic and calcium channel blocking activity.[1,2] Although almost all effective antiarrhythmic drugs are implicated in causing proarrhythmic effects, propafenone

and other class IC drugs (such as flecainide) have greater potential for triggering sustained ventricular tachycardia compared to IA or IB drugs. Propafenone in particular has 5-10% overall incidence of proarrhythmia[3,4] and is prone to causing proarrhythmic effects in patients with underlying structural heart disease by slowing conduction and creating unfavourable imbalance between conduction and refractoriness.[5] Propafenone is considered to be among first-line treatments for conversion of recent onset atrial fibrillation but is contraindicated in patients with congestive heart failure, severe systolic dysfunction, sinoatrial / atrioventricular / intraventricular disorders of impulse conduction or sinus node dysfunction, patients with unstable angina, a recent or acute myocardial infarction.[6] Caution should be taken for all these conditions to avoid incessant ventricular tachycardia, dramatic decrease in cardiac output and circulatory arrest.

REFERENCES

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Cardiology News /Recent Literature Review

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The **ESC** Annual Congress is slated for 27-31/8/2011 in Paris

The **TCT** Annual Conference will be held in San Francisco 7-11/11/2011

The **AHA** Annual Scientific Sessions are scheduled for 12-16/11/2011 in Orlando

The **Athens Cardiology Update 2012** is slated for April 5-7, 2012

Reverse remodeling in cardiac resynchronization therapy reduces the risk of ventricular tachyarrhythmias in the MADIT-CRT trial

The risk for ventricular tachyarrhythmias (VTA) (including ventricular tachycardia, ventricular fibrillation and ventricular flutter) was assessed in patients receiving cardiac resynchronization defribrillator therapy (CRT-D) or cardioverter-defribillator therapy (ICD) according to echocardiographic findings during 1-year follow-up. It was found that high responders to CRT-D (defined as ≥ 25% reduction in left ventricular end-systolic volume) experience a significant 55% reduction in the risk of VTA compared with ICD only patients ($p<0.001$), whereas the risk of VTA was not significantly different between low responders and ICD-only patients ($p=0.21$). (Barsheshet A et al, *J Am Coll Cardiol* 2011; 57: 2416-2423)

High-dose atorvastatin does not prevent development of atrial fibrillation in patients with prior stroke or transient ischemic attack in the SPARCL trial

Statins are included as upstream therapy for prevention of new-onset of atrial fibrillation (AF) in the 2010 guidelines for the management of AF. The SPARCL trial tested the hypothesis that long-term treatment with high-dose atorvastatin (80 mg) reduces the occurrence of AF in 4731 patients with prior stroke or transient ischemic attack who were followed up for a median of 4.8 years (patients with prior paroxysmal AF were excluded from the trial). It was concluded that the time from randomization to first occurrence of new AF did not differ between atorvastatin (139 cases of new AF) and placebo (122 cases) group (hazard ratio 1.15, 95% CI 0.90-1.46, P=0.26). (Schwartz GG et al, *Am Heart J* 2011; 161: 993-999)

Frequent ventricular pauses in patients receiving ticagrelor in the acute phase of acute coronary syndromes, according to a sub-analysis of the PLATO study

A prospective continuous electrocardiographic assessment was performed within the PLATO study comparing ticagrelor and clopidogrel in 2908 patients hospitalized with acute coronary syndromes. During the first week, ventricular pauses (≥ 3 sec) occurred more frequently in patients receiving ticagrelor (relative risk 1.61, p=0.006) but at 1 month pauses occurred less frequently and treatments were similar between groups. It is suggested that there are no apparent clinical consequences related to the excess in ventricular pauses in patients assigned to ticagrelor while the incidence of clinical adverse events (including syncope, pacemaker placement and cardiac arrest) was no different (Scirica BM et al, *J Am Coll Cardiol* 2011; 57: 1908-1916).