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Case Report Open Access

Flecainide Induced Ventricular Arrhythmias in Atrial Fibrillation

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Abstract

Purpose: Flecainide is a class 1C antiarrhythmic drug especially used for the management of supraventricular arrhythmia. Flecainide also has a recognized proarrhythmic effect in patients treated for ventricular tachycardia. It is used to treat a variety of cardiac arrhythmias including paroxysmal fibrillation, Paroxysmal Supraventricular tachycardia and ventricular tachycardia. Flecainide works by regulating the flow of sodium in the heart, causing prolongation of the cardiac action potential. The proarrhythmic effects however noted are not widely reported.

Method: We report a case of paroxysmal atrial fibrillation with structurally normal heart who was treated with oral Flecainide. Despite subjective improvement and no adverse events [QTc prolongation] a repeat holter detected him to have multiple short non sustained ventricular arrhythmias.

Results: Development of ventricular arrhythmias, salvos & non sustained ventricular tachycardia after a month of initiation of oral Flecainide detected by 24 hours ECG holter lead to discontinuation of Flecainide and subsequent early electro physiological studies and successful ablation.

Conclusion: Initiation of oral Flecainide in a case of atrial fibrillation with subjective improvement and regular ECG monitoring, no QTc prolongation can still lead to development of dangerous ventricular arrhythmias. A cautious approach and thorough investigations and follow up are recommended.

Introduction

Flecainide is a class 1C antiarrhythmic drug used especially for the management of supraventricular arrhythmias like atrial fibrillation (AF) [1]. It causes rate- dependent slowing of the rapid sodium channel slowing phase 0 of depolarization and in high doses inhibits the slow calcium channel [2]. Flecainide also slows conduction in all cardiac fibers, increasing conduction times in the atria, ventricles, atrio-ventricular node and His-Purkinje system. Flecainide can also cause myocardial depression. In over- dose cases, flecainide can induce life treating ventricular arrhythmias and cardiogenic shock.

Case Report

Mr. RJN, 44 years male was diagnosed with paroxysmal atrial fibrillation in May 2013 and was under beta blockers and acetyl salicylic acid. He was reviewed in our hospital in September 2013 because of his disturbing symptoms of palpitations and fatigue. Beta blockers were stopped and he was started with flecainide and dabigatran with the possibility of electrical cardioversion later if required. Regular follow ups were done and he reported subjective improvement starting after 3 days. Periodic ECG done did not show any QTc prolongation. He was reassessed with holter after one month of Flecainide treatment and found to have multiple short episodes of ventricular arrhythmias [salvos and non-sustained ventricular tachycardia] while still remaining in paroxysms of atrial fibrillation. Thereafter he was admitted to CCU and flecainide was stopped. He was switched back to Beta blockers and again reassessed with holter after a week which showed persistent atrial fibrillation with no ventricular tachyarrhythmia.

Risk profile

No hypertension or diabetes. Nonsmoker

Physical examination

BP, 110/70 mm of Hg, PR, 102/minute irregular. No evidence of heart failure.

ECG: Initial

Atrial fibrillation, rate ~110/minute. AF currently

With flecainide

Paroxysmal AF with multiple non sustained ventricular arrhythmias.

ECHO

Atrial fibrillation, Normal LV dimensions and systolic function.

He underwent electrophysiological studies and was successful isolation of all four pulmonary veins for paroxysmal atrial fibrillation with termination of focal site for AF initiation near mid/proximal coronary sinus roof.

Discussion

Pharmacological treatment for atrial fibrillation

Antiarrhythmic drugs used for pharmacological cardioversion of AF include disopyramide, procainamide, quinidine, flecainide, propafenone (both class IC), dofetilide, ibutilide, sotalol, and amiodarone (all class III). However the most commonly used drugs for chemical cardioversion are flecainide, sotalol, and amiodarone. In patients with new onset AF, successful cardioversion is reported in approximately 80% of cases with oral therapy, increasing up to 90% with intravenous administration [1].

Unfortunately however the recurrence of AF is common and frequently requires long-term medications to improve maintenance

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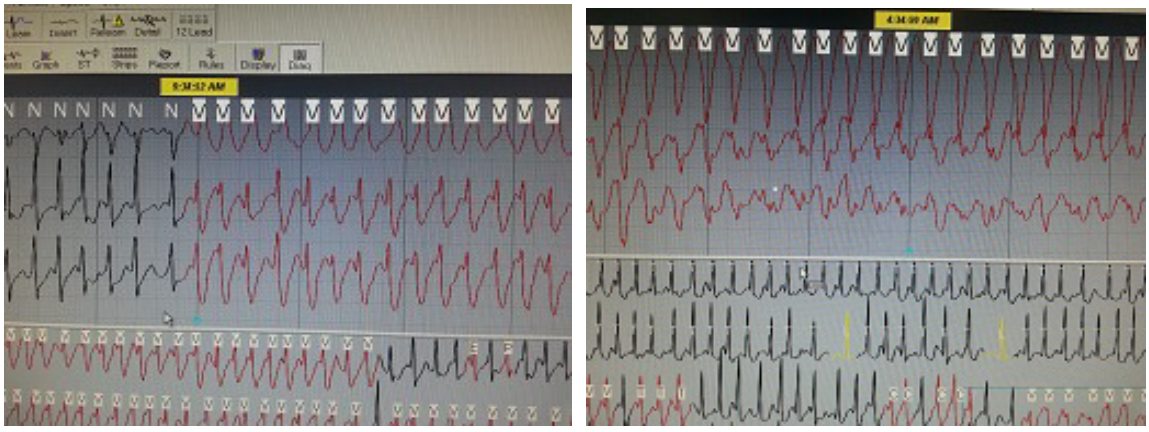

Figure 1: ECG Holter showing ventricular arrhythmias.

of sinus rhythm. For most commonly used agents, the relapse rate is around 50% during the first year, although slightly better figures are seen with dofetilide and amiodarone [2-8]. Many studies have demonstrated that flecainide and propafenone are effective drugs for preventing AF recurrence [9-11]. Flecainide is superior to quinidine with regards to fewer side effects [12]. In contrast, propafenone is more effective for maintenance of sinus rhythm than quinidine, and as effective as sotalol [13,14]. Class IC drugs are usually preferred to class IA drugs in view of their better safety profile. [12,13]. The success of electrical cardioversion for AF has been quoted as between 75 and 93%, although this depends on left atrial size and co-existing structural heart disease, and ultimately on the duration of AF [15-17]. Amiodarone or sotalol can be used pre-cardioversion to improve the success of electrical cardioversion [18]. Such an approach is advocated by the ACC/AHA/ESC guidelines on AF management [2]. The frequency of recurrence of AF after electrical cardioversion is high, and maintenance therapy with antiarrhythmic drugs such as amiodarone or sometimes b-blockers is somewhat useful to prevent AF relapses [1]. B-blockers are very effective at controlling ventricular rate and also may reduce the risk of AF recurrence following successful cardioversion (whether spontaneous, pharmacological, or electrical) and are currently used as first-line prophylactic agents in paroxysmal AF. B-blockers have also been shown to reduce the frequency of post-operative AF, although sotalol (which also has class III effects) appears to be the most effective in this setting. As AF commonly coexists with hyper- tension or congestive heart failure, b-blockers may also be part of conventional therapy in such patients. Rate-limiting, non-dihydropyridine calcium channel blockers (diltiazem, verapamil) are frequently used to optimize rate control where b-blockers are contraindicated or ineffective. An intravenous B-blocker (for example, esmolol or metoprolol) or rate-limiting calcium antagonists (diltiazem, verapamil) are indicated where urgent pharmacological rate control is required. Intravenous amiodarone is a useful alternative in situations where the administration of b-blockers or calcium antagonists is not feasible, such as in the presence of heart failure. All current class IA, IC, and III antiarrhythmic drugs have significant side effects. This includes non-cardiovascular effects (e.g. pulmonary fibrosis and thyroid dysfunction with amiodarone), and of particular importance, the risk of life-threatening ventricular proarrhythmia including TdP in up to 5% of patients [19,20]. Most of these antiarrhythmic drugs prevent or terminate AF by altering the function of potassium or sodium channels within the atrial cells. Blockade of potassium channels may prolong ventricular repolarization and hence, the refractory period-resulting in QT-interval prolongation. Given the risk of severe proarrhythmia, the safety profile of many current antiarrhythmic drugs is far from ideal. Amongst the most worrying side effects are QT-interval prolongation and risk of proarrhythmia, including torsade de pointes (TdP) [21].

Flecainide, a class 1C anti-arrhythmic agent, depresses the rate of depolarization of cardiac action potentials producing a membrane stabilizing action. It is a very effective anti-arrhythmic agent against supraventricular arrhythmias; nevertheless flecainide is contraindicated in patients with structural heart disease because it increased mortality [22]. The proarrhythmic effect of flecainide may be related to promoting a reentry in ventricular tissue. The phenomenon is due to a rate-dependent blockade of rapid sodium channels slowing phase 0 of depolarization and an inhibition of the slow calcium channel [23]. In cases of overdose, the mortality with class IC agents has been reported to approach 22%. Conduction disturbances began with widening of QRS complex which can rapidly progress to ventricular tachycardia, electromechanical dissociation and asystole.

Despite the large number of available antiarrhythmic agents, significant QT-interval prolongation and risk of severe proarrhythmia, including torsade de pointes, limit pharmacological opportunities in the management of atrial arrhythmias.

Future

In conclusion, despite the large number of antiarrhythmic agents that are currently available, modern cardiology is still waiting for the introduction of new efficient and safe drugs for the treatment of atrial arrhythmias. The ideal anti- arrhythmic agents must efficiently cardiovert AF patients and prevent relapses without proarrhythmic potential. To achieve this, it seems that such drugs should be atrial selective, should have multi ion-channel effects, should not increase transmural dispersion of repolarization, should not produce early after depolarization , and should not exhibit reverse use-dependency.

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