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Case Report

Case Report of Oral Sotalol on Atrial Fibrillation Conversion and Supraventricular Tachycardia Cardiac Arrhythmia Control

Howard B Reinfeld*

Cardiology Graduate of Mount Sinai Hospital Miami Beach Florida and Cardiologist at Private Practice, Howard B Reinfeld and Associates MD, PA and BS Pharmacy, St. John's University, Jamaica, New York, USA

***Corresponding Author:** Howard B Reinfeld, Cardiology Graduate of Mount Sinai Hospital Miami Beach Florida and Cardiologist at Private Practice, Howard B Reinfeld and Associates MD, PA and BS Pharmacy, St. John's University, Jamaica, New York, USA.

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Abstract

Sotalol is a potassium channel inhibitor that also functions as a non-cardio selective beta-blocker. It is a class III agent under the Vaughan-Williams categorization system for antiarrhythmic drugs. It primarily blocks potassium channels. Indications for sotalol include supraventricular tachycardia, hemodynamically stabilized ventricular irregular heartbeat, pharmaceutical cardioversion of atrial fibrillation, preserving sinus rhythm, and surgical atrial fibrillation after heart surgery.

Keywords: Potassium channel inhibitor; Sotalol; Antiarrhythmic drugs.

Introduction

Atrial fibrillation and SVT are similar and yet different in their own way. The risk factors for atrial fibrillation may include heart failure, heart disease, alcohol, heart attack, sleep apnea, obesity, diabetes, and certain medications. However, SVT is more likely to affect women than males. People with anxiety, a lot of caffeine, smoke, and alcohol use are often at risk of SVT [1]. When it comes to the symptoms of AFib and SVT, both share similar symptoms, such as chest tightness, heart palpitations, fatigue, mild pain, irregular heartbeat, mild shortness of breath, lightheadedness, and even fainting in some cases. Their severe symptoms also appear as chest pain and trouble breathing.

The diagnosis of both atrial fibrillation and Supraventricular tachycardia can be performed through an electrocardiogram (EKG) [2]. Through this test, the electrical activity of the heart is measured. The heart activity is examined through an EKG device by wearing a Holter monitor for two to three

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days. Smartwatches are also available for heart rhythm monitoring [2]. If a person is diagnosed with SVT or AFib, he might get treated with oral sotalol. It is a group of medicines known as beta blockers which are used to treat AFib, SVT, and many other arrhythmia conditions.

Mechanism of action

Sotalol is a potassium channel inhibitor that also functions as a non-cardio selective beta-blocker. The action potential length and beneficial refractory duration in the atrial, ventricular, nodal, and extra nodal tissue are both prolonged by sotalol. It effectively inhibits potassium current through competition. The highest potassium prevailing blocking function of sotalol happens when the cardiac rate is low, raising the possibility of QT lengthening and torsade's de pointes under bradycardia situations. Sotalol demonstrates reversal user-dependent actions. It only takes a small amount to have a beta-blocking action. For instance, a good beta-blocking activity might be 25 mg but requires a higher dose to have class III antiarrhythmic effects, which are around 80 mg. The fast delayed-rectifier K+ current, IKr, which is essential for appropriate repolarization of the heart's ventricles, is mediated by potassium channels that are encoded by the human Ether-à-go-go-Related (HERG) gene, according to a recent study. As a result, HERG is a pharmaceutical priority for antiarrhythmic medicines in classes Ia and III [3].

Voltage-dependent K+ channel blockers (Class IIIa), both nonselective (amiodarone) and selective ones (HERG; sotalol), are now included in Class III agents. Blockers of the Kv11.1 (HERG) channel that regulate the swift K+ current (IKr) cause a delay in the recovery of the atrial, Purkinje fibers, and ventricular myocyte action potentials as well as an increase in the effective refractory period (ERP) and the amount of time it takes for the AP to recover, all of which reduce the re-entrant tendency [4]. Sotalol's simultaneous propensity for effectively inhibiting receptors without preference for j3i and the ability to lengthen the cardiac action prospective duration are what drive the drug's overall effect (APD). Sotalol is a racemate of d-isomers and 1-isomers, each of which shows an equivalent class III effect; only the 1-isomer exhibits considerable 3-adrenoceptor-blocking activity. Its structural formula is 4'-(2 isopropylamino-1-hydroxyethyl) methylsulfonamide [5].

Beta-blocker activities

Sotalol is a nonselective-adrenoceptor blocker that has negligible to no effects on sodium channels and no detectable intrinsic sympathomimetic effect. In both in vitro and in vivo testing paradigms, sotalol results in competitive blockage of both B1 and B2 adrenoceptors [6]. Such a reaction is the result of repolarization becoming longer, which allows calcium to enter the myocyte for longer periods of time throughout each cardiac cycle.

Significant medical consequences stem from this result. In addition, it is anticipated to reduce the potential decline in ventricular activity that could occur in individuals who already have myocardial infarction-related damage (3-blockade) (MI). This anticipation

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is in accordance with the finding that sotalol exerts slightly less adverse inotropic effects than the majority of other 3-blockers.

Dosage of sotalol for AFib and SVT

Both oral and intravenous versions of sotalol are available. The dose of sotalol administered intravenously vs orally can be converted. 80 mg of sotalol taken orally is comparable to 75 mg given intravenously, and 160 mg is equivalent to 150 mg given intravenously. It is advised to deliver the medication intravenously slowly because a fast infusion might result in hypotension [7]. Depending on renal function, 120 mg twice daily is the recommended dose to prevent STV and atrial fibrillation. Different metrics, such as lengthening the QT interval, sinus cycle duration, atrial and right ventricular effective refractory periods, and atrioventricular node relative and functional refractory periods, do not significantly vary between intravenous and oral sotalol.

Interactions of sotalol

There are many drugs that should not be taken with few other medications as they might interact and cause complications for overall health. In the sotalol case, fingolimod is a drug that may interact with this product [7].

Along with sotalol, other medications such as disopyramide, amiodarone, dofetilide, procainamide, pimozide, quinidine, and macrolide antibiotics can also influence the cardiac rhythm (QT prolongation in the EKG). Several products include components that are likely to increase the heart rate. It is better to discuss with the pharmacist or physicians about the medications before taking them.

Role in atrial fibrillation conversion

The most prevalent persistent cardiac arrhythmia in medical practice is atrial fibrillation (AF), and its frequency rises with advancing age [8]. AF can develop in people without medical or instrumental indications of cardiopulmonary disease (hence called "lone" AF) [9], or it can be linked with other heart disorders such as congestive cardiac failure, hypertension, valvular disease, and myocardial infarction [10]. An AAD possessing class II (b-adrenoceptor inhibiting) and class III (prolonging the length of the cardiac action potential) features is exhibited by sotalol. This medicine is an intriguing choice for the management and control of AF due to its unusual property. Despite a relatively high risk of relapses (about 50%) after 1-year follow-up, identical studies have shown that sotalol has equivalent effectiveness in preventing relapses of AF as propafenone and flecainide [11,12]. Manz M, et al. studied the outcomes of intravenous sotalol (80 mg) in nine patients with AV nodal re-entrant PSVT and 11 patients with Wolff-Parkinson White syndrome [13].

Sotalol delayed transmission in the AV node and bypass routes in both the anterograde and retrograde directions, lengthening the right atrium and ventricle's operative refractory period. The ventricular rhythm during AF was shown to range between 148 and 112 beats/min, and the tachycardia rate was reduced from 182 to 153 beats/min. All areas of the reentrant network, such as the atrial, ventricular, AV node, and bypass

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routes, were depressed by sotalol. Accordingly, the data suggest that the medication may be beneficial in avoiding both reciprocating tachycardias and the speedy ventricular reaction to AF in individuals with Wolff-Parkinson-White syndrome. The benefits of sotalol were assessed during electrophysiologic testing as well as long-term preventive medication in research [14] of 22 individuals with the Wolff-Parkinson-White syndrome who were resistant to numerous conventional drugs. 13 out of 18 individuals with inducible tachycardia had their persistent reciprocating tachycardia made non-inducible during the electrophysiologic assessment. The auxiliary routes greatly extended their anterograde and retrograde active durations by 20% and 28%, respectively.

A follow-up duration of 1 to 47 months demonstrated 77% effectiveness in managing symptomatic tachycardia after long-term medication. Cardiac positive benefits of the medication should be taken into account in light of newly available information on electrode catheter ablation of all these arrhythmias, a procedure that is extremely efficient in curing reentrant PSVT with or without bypass passages in the majority of instances.

Pharmacodynamics

A powerful non-cardio selective b-adrenoceptor blocker without inherent sympathomimetic or membrane-stabilizing effects is sotalol hydrochloride [15]. Sotalol extends the QT gap on the electrocardiogram and lengthens the period of action possibilities observed in cardiac tissue, in contrast to other b-adrenoceptor-inhibiting medications [16,17]. Sotalol is a medication that possesses both class III (cardiac action prospective period extension) and class II (b-adrenoceptor inhibition) antiarrhythmic characteristics. In order to establish an adrenergic blockade, sotalol must be administered at higher doses [18,19] and only at a daily dosage of 160 mg and beyond are substantial class III effects observed.

Adverse effects

The side effect profile of sotalol is innate to its modes of action as a non-cardio selective beta-blocker and a potassium-channel blocker. The QTc is prolonged by the potassium channel blockage, especially in phase 3 of the cardiac action potential. As a result, monitoring the ECG is necessary while starting sotalol or adding additional QTc-prolonging drugs. In 1% to 2% of instances, the QT interval lengthens, which might result in torsades de pointes, ventricular fibrillation, or new ventricular tachycardia. Sotalol's adverse effect QT-prolonging impact is closely correlated with its serum level. The risk of torsades is 1% at dosages below 320 mg and rises to 5% at doses over 320 mg.

Its negative effects, particularly torsades de pointes, are likewise dose dependent. If a patient has an implanted cardioverter-defibrillator, higher dosages may be used. With the IV formulation, the frequency of QT prolongation is more common. Its non-cardio selective beta-blockade also has bradycardia, dyspnea, exhaustion, and increasing heart failure as side effects. A life-threatening ventricular tachycardia linked to QT prolongation can be caused by sotalol.

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Research has shown that it is necessary to reduce the dose, prolong the time between doses, or stop the medication intake if the QT interval is less than 500 msec. Essentially, calculate creatinine clearance prior to using sotalol to establish the proper dosage.

Different drug isomers may have different pro-arrhythmia risks. For instance, the antiarrhythmic medication sotalol has d-and l-enantiomers. According to a recent study that used molecular dynamics, d-sotalol had a larger pro-arrhythmia risk than l-sotalol [1]. According to the creatinine clearance, the beginning dose for intravenous sotalol is 75 mg administered over 5 hours once or two times. Over a five-hour period, this dosage can be increased up to 112.5 mg. The initial dosage is the same for ventricular arrhythmias, and this can be raised by 75 mg/day every three days. There have been oral dosages of as much as 240–320 mg, which is equivalent to 225–300 mg administered intravenously. 120 mg od or bd was the most beneficial dose for preventing AF (equivalent to 112.5 mg IV). However, dosages as high as 150 mg IV or 160 mg od or bd have been applied. According to the 2015 American Heart Association (AHA) protocols for cardiopulmonary resuscitation (CPR) and emergency cardiac care, intravenous sotalol can be used to treat broad complicated tachycardias [6].

Prevention of AF

Sotalol could be used to avoid AF in spite of its limited ability to turn AF into SR. Numerous studies have studied the effectiveness of oral medicine delivery for individuals with paroxysmal AF or recurrence of AF following successful CV in preventing arrhythmia recurrence. Sotalol was reported to be safe and efficacious at doses varying from 80 to 160 mg two times daily in two placebo-controlled studies including patients in SR with at least one known past episode of AF [20,21]. 253 individuals who had a background of symptomatic confirmed AF or atrial flutter during the preceding three months and were in SR at the time of randomization were given sotalol at various doses or with a placebo in the research by [22]. With a dose-response relationship in lowering the incidence of symptomatic relapses, the relapse-free survival likelihood at one year for the placebo group, the 80-, 120-, and 160-mg sotalol everyday-dose groupings was 28, 30, 40, and 45%, respectively [19].

In the other small research in individuals with paroxysmal AF, a similar dose requirement in medical effectiveness was demonstrated [20]. Recent randomized controlled research comparing the time to AF recurrence observed by trans telephonic monitoring following effective CV in patients with chronic and long-lasting AF managed with amiodarone, sotalol, or placebo [21] demonstrated that sotalol is superior to placebo in rhythm management. Among the 261 patients who received sotalol treatment, the median period until AF relapse was 209 days as opposed to 13 days in the control group [23].

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potassium channel blockage, especially in phase 3 of the cardiac cycle. As a result, monitoring the ECG is necessary while starting sotalol or adding additional QTc-prolonging drugs. In 1% to 2% of cases, the QT interval lengthens, which might result in torsade's de pointes, new ventricular tachycardia, or ventricular fibrillation. Sotalol's unfavorable QT-prolonging effect is directly correlated with its serum level [24]. The rate of torsade's is 1% at dosages below 320 mg and rises to 5% at doses over 320 mg. Its negative effects, particularly torsade's de pointes, are likewise dose dependent. If a person has an implanted cardioverter-defibrillator, higher doses may be used. With the IV formulation, the occurrence of QT delay is more common. Its non-cardio selective beta-blockade also has bradycardia, exhaustion, dyspnea, and increases heart failure as side effects.

Case 1

Patient is a 80 y/o F with h/o atrial fibrillation, paroxysmal SVT, obstructive sleep apnea, CAD, ASHD, renal insufficiency, palpitations, carotid stenosis 30-40%, cardiac cataracts, HTN and hyperlipidemia. This patient was treated with Sotalol 80mg twice a day and well controlled with oral sotalol.

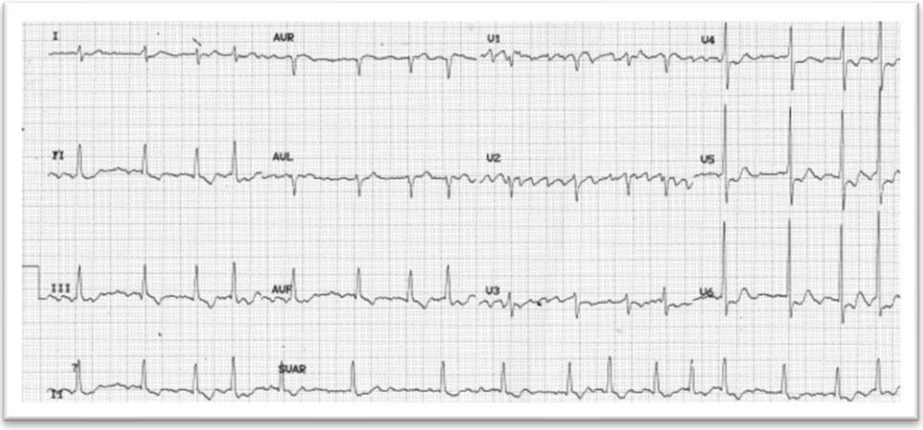

Figure 1: Afib EKG prior to oral sotalol treatment.

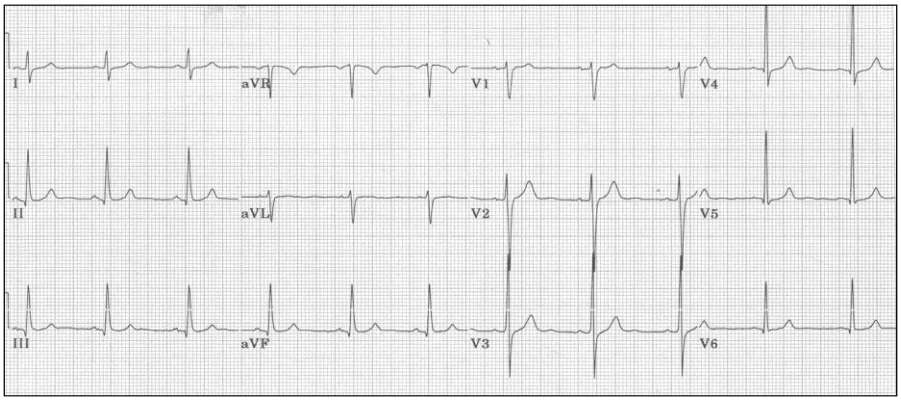

Figure 2: Post oral sotalol EKG showing AFIB conversion to normal sinus rhythm.

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Case 2

Patient is a 43 y/o M with no significant PMHx presents to the office after being referred to my office due to chest pain, palpitation, and hyperlipidemia.

A cardiac ischemic workup was conducted and was negative. An echocardiogram was performed with a normal expression. Upon the review of his 24hour monitor findings concluded multiform ventricular premature complexes and NSVT. The patient was started on 40 mg of oral sotalol twice a day and monitored with a 30-day event recorder which showed significant rate control with normal sinus rhythm. At the time of reassessment, the patient also stated his symptoms have resided.

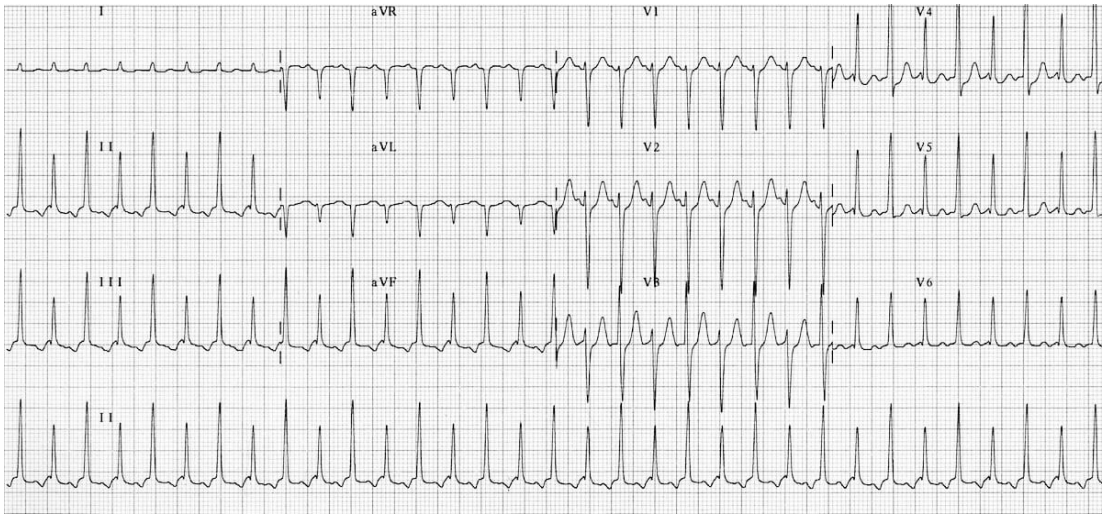

Figure 3: NSVT prior to oral sotalol treatment.

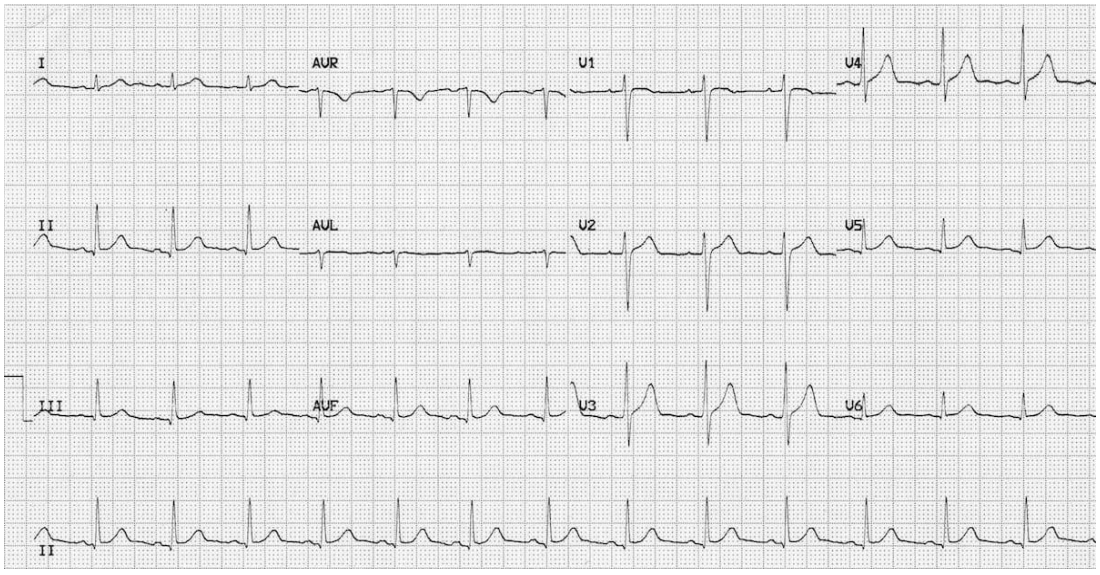

Figure 4: EKG post sotalol treatment in sinus rhythm.

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Conclusion

The beta-adrenergic blocking medication sotalol has no inherent sympathomimetic action. Additionally, it has the unusual ability to produce a noticeable lengthening of the cardiac activity current duration. Sotalol capacity to extend refractoriness in the node and extra node tissue underlies its antiarrhythmic effects. Therefore, individuals may not end up benefiting from its anti-arrhythmic capacity when given a lesser dose over an extended period of time. Atrial fibrillation and Supraventricular tachycardia have so much in common, including symptoms, diagnosis, and treatment. Both are cardiac arrhythmias and can lead to severe complications and even fainting if not treated properly. Sotalol is one of the most effective drugs used to treat Atrial fibrillation and Supraventricular tachycardia.

It has the tendency to impact in a better way when taken orally in the right dosage. However, it is also available intravenous. Higher quantities and over-dosage of sotalol may lead to adverse effects and can become life-threatening. Hence, it is often recommended to take the prescribed dosage from professionals to treat AFib and STV.

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