

# A Classical Wide Complex Tachycardia due to Propafenone Toxicity; Uncommon Side Effect of a Commonly Used Medication

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**Abstract** Propafenone is a Class 1 C anti-arrhythmic drug, frequently used in the treatment of supraventricular and ventricular arrhythmias. We herein are presenting a case of 67-year-old female who presented with wide complex tachycardia consistent with Propafenone toxicity and who responded immediately to sodium bicarbonate injections. This case shines the light on a common effect of Propafenone toxicity and aims to increase the clinician awareness about such a documented reaction, that can be reversed by a simple treatment like sodium bicarbonate.

**Keywords:** propafenone, tachycardia, atrial fibrillation

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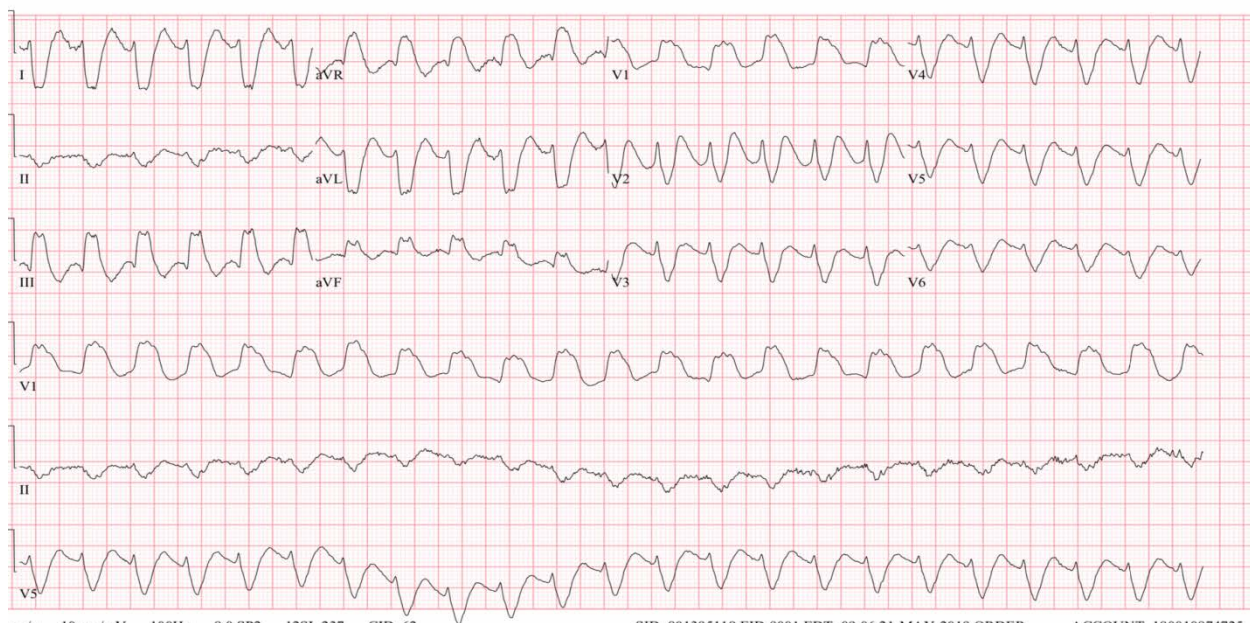
## 1. Introduction

Atrial fibrillation is a very commonly managed with either rate control or rhythm control medications. Propafenone is frequently used in the treatment of supraventricular and ventricular arrhythmias [1]. It is a Class 1 C anti-arrhythmic drug under the Vaughan Williams classification. It also has beta-receptor, sodium channel and calcium channel antagonist activity [2]. It is metabolized through cytochrome P450 2D6 system in the liver; the major metabolites (5-hydroxypropafenone and N-despropyl Propafenone) express that anti-arrhythmic activity. Serious side effects especially when in toxic levels can lead to prolonged QT interval which progresses to ventricular tachycardia. We describe a case of Propafenone toxicity that presented with wide complex tachycardia and resolved after administration of intravenous sodium bicarbonate.

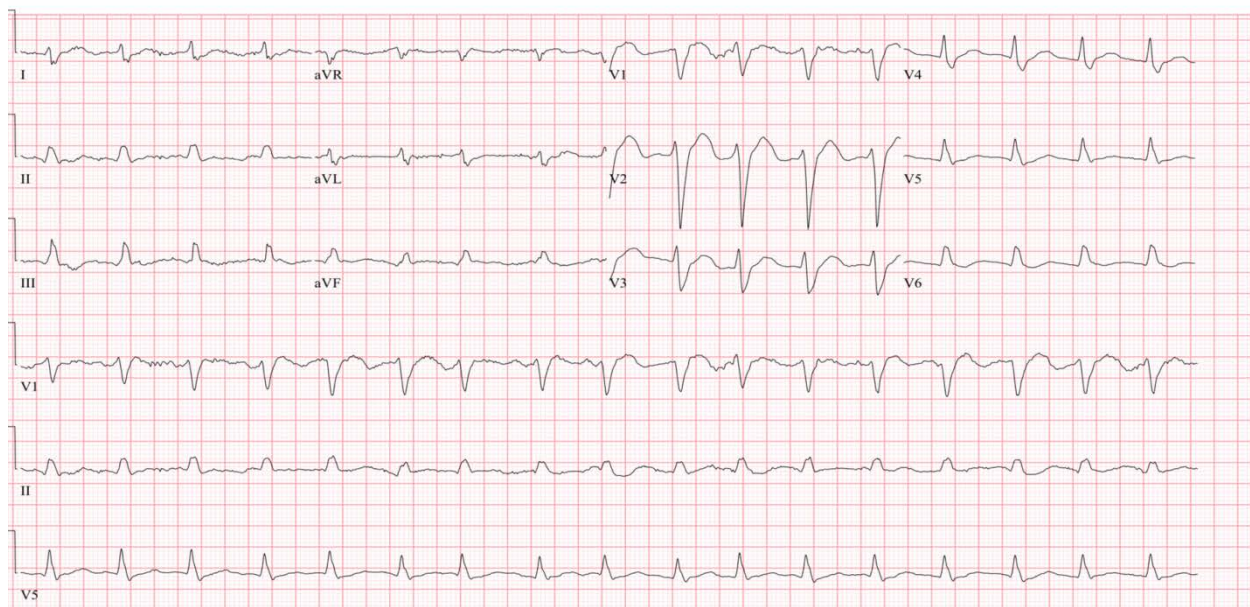
## 2. Case Presentation

A 67-year-old Caucasian female with past medical history of permanent atrial fibrillation on Propafenone with Watchman device placed, morbid obesity, hypertension, obstructive sleep apnea on CPAP, osteoarthritis, presented with complaints of right pleuritic chest pain with no radiation for a couple

days. She reported feeling dizzy, fatigued and dyspneic for about 1 week and at the day of admission her lips went numb and she was extremely fatigued. She denied fever, syncope, diarrhea, nausea, vomiting, trauma, illicit drugs abuse, any past history of diabetes, GERD or GI ulcers. Denied any recent illness or URI symptoms. In the emergency room the patient was hypotensive, and initial labs obtained were within normal limits including troponin. EKG showed wide complex tachycardia concerning for Propafenone toxicity (Figure 1). Patient received a 50 mEq dose of intravenous sodium bicarbonate with repeat EKG showing complete resolution of her symptoms and converting of her rhythm to atrial flutter with aberrancy (Figure 2). Patient was admitted to the CCU for observation overnight. Her thyroid function tests were within normal and a urine drug screen was negative. She was not on any other medications that can cause tachycardia/arrhythmia. She remained hemodynamically stable and asymptomatic. Her Propafenone was discontinued and she was started on Metoprolol tartrate 25 mg twice daily. Her EKGs showed normal sinus rhythm with heart rate of 60s-50s. Echocardiogram was done which revealed, good left ventricular systolic function. Visually estimated left ventricular ejection fraction is approximately 55%. The Watchman device appears coaxially seated and stable. Left heart catheterization was done which was normal. Before discharge, the patient underwent electrophysiological study and atrial flutter ablation. She was discharged home with appropriate follow-up.



**Figure 1.** EKG shows wide complex tachycardia at presentation consistent with Propafenone toxicity.



**Figure 2.** EKG shows underlying atrial flutter with 2:1 block and aberrancy after intravenous administration of 50 mEq of sodium bicarbonate

### 3. Discussion

Propafenone is a class IC antiarrhythmic with effects on the sodium channel. It has pro-arrhythmic and myocardial inhibitory actions that can affect patients with low ejection fraction. Accidental or iatrogenic ingestion of high dose of Propafenone or any medication that interacts with metabolism of Propafenone can precipitate lethal toxicity [2]. Propafenone toxicity is directly dependent on blood levels [3]. Multiple complications such as hypotension, bradycardia, widened QRS, and even cardiac arrest can be observed after severe Propafenone toxicity [2]. In one study, the incidence of first-degree heart block and bradycardia with Propafenone was 1-3% [2]. However, prolongation of PR interval is an accepted side effect of sodium channel blockers in view of prolongation of action potential. Propafenone has been seen to prolong QRS

duration and can lead to rate-dependent QT prolongation [2], as well as left bundle branch block [4].

Pro-arrhythmic risk is very high in patients with coronary artery disease and with patients with ventricular scars [5]. Other factors might also increase Propafenone toxicity like alcoholism and other QT prolongation medications. In one case, Propafenone toxicity was reported after binge drinking of alcohol, where the patient presented with wide QRS and first-degree heart block that improved after a sodium bicarbonate infusion [6].

Treatment is usually reversal of cardiac depressive beta blocking effect with glucagon, and sodium bicarbonate ( $\text{NaHCO}_3$ ) is the antidote of choice. It interacts with sodium channels and displaces Propafenone.  $\text{NaHCO}_3$  is a very rapid acting medication and effects can be demonstrated electrocardiographically in minutes [7]. Thus, hypertonic sodium bicarbonate should be infused as

soon as Propafenone toxicity is suspected. Hypertonic saline and temporary pacing might be required in hemodynamically unstable patients [7].

In general, the higher the blood level of Propafenone, the worse is the prognosis, especially in the elderly population and patients with structural heart disease. However, not all Propafenone toxicity cases respond as expected to standard medical therapy. Jkovic et. al. reported a case of Propafenone toxicity in a patient who presented with severe bradycardia with wide QRS who did not respond to sodium bicarbonate, pacing and inotropic support either, reflecting the lethality of Propafenone-related conduction side effects [8].

## 4. Conclusion

Our patient was on Propafenone and presented with the classical wide QRS complexes expected to be seen with Propafenone toxicity and responded to a single dose of intravenous sodium bicarbonate. Our case highlights the clinical importance of recognizing and promptly treating pro-arrhythmic complications of a commonly utilized antiarrhythmic agent.

## Conflict of Interest

None of the authors have any conflicts of interest to declare.

## Disclosure of Funding

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