Malign Arrhytmia Development Due to Propafenone Over Dose: a Case Report

Propafenon Aşırı Dozuna Bağlı Gelişen Malign Aritmi: Olgu Sunumu

Ethem ACAR,[1] Latif DURAN,[2] Yahya ŞAHİN,[2] Hızır Ufuk AKDEMİR,[2] Mehmet EKİZ,[2] Ali Kemal ERENLER[3]

[1]Department of Emergency Medicine, Erzurum Region Training and Research Hospital, Erzurum;
[2]Department of Emergency Medicine, Ondokuz Mayıs University Faculty of Medicine, Samsun;
[3]Department of Emergency Medicine, Corum State Hospital, Corum, all in Turkey

SUMMARY

Propafenone is a group 1C antiarrhytmic agent. It is an agent used in patients with symptomatic supraventricular tachycardia requiring treatment, such as atrioventricular node tachycardia, Wolff-Parkinson-White Syndrome and paroxysmal atrial fibrillation. It is also used in life-threatining symptomatic ventricular tachycardia requiring treatment and with excessive intake it has serious side effects on cardiovascular, gastrointestinal, nervous, haematological and dermatological systems. In this report, we present a case of propafenone intake with suicidal purpose and we aim to share our experience with malign arrhytmia development and arrhytmia management. A 22-year-old female patient presented to our emergency department with complaints of general situation distortion and feeling sick after ingesting 20 pills (6 g) of her friend's Propafenone HCl 300 mg for suicidal purpose one hour previously. In the electrocardiography (ECG), regular rhythm, wide QRS and the absence of P-wave was observed. Pulseless ventricular tachycardia developed and defibrillation with 360 joule was performed followed by cardiopulmonary resuscitation. $NaHCO_3$ administration of 1 mEq/kg every 4 hours was initiated. After the therapy, QRS duration shortened. The patient became conscious with spontaneous ventilation. Early diagnosis and appropriate resuscitative interventions can be vital in propafenone intoxication. $NaHCO_3$ administration in the presence of hypotension and ECG abnormalities are vital.

Key words: Intoxication; malign arrhytmia; prorafenon.

ÖZET

Propafenon grup 1C antiaritmik ajandır. Atriyoventrikül düğümü taşikardileri, Wolff-Parkinson-White sendromu veya paroksismal atriyal fibrilasyonu olan hastalardaki supraventriküler taşikardiler gibi tedavi gerektiren semptomatik supraventriküler taşiaritmiler ve tedavi gereği görülen, yaşamı tehdit eden semptomatik ventriküler taşiaritmilerde kullanılan; yüksek doz alımında kardiovasküler sistem, gastrointestinal sistem, sinir sistemi, hematolojik sistem ve deri üzerinde ciddi yan etkisi olan bir ilaçtır. Bu yazıda, intihar amaçlı propafenon alan bir olguyu sunarak, malign aritmi gelişimi ve yönetimi hakkındaki deneyimimizi paylaşmak istedik. Yirmi iki yaşında kadın hasta yaklaşık 1 saat önce arkadaşının propafenon HCL 300 mg'lık ilacından intihar amaçlı 20 tb (6 g) aldıktan sonra genel durum bozukluğu şikayeti ile acil servisimize başvurdu. Çekilen elektrokardiografisinde (ECG) ritmin düzenli, QRS'in geniş ve P dalgasının olmadığı görüldü. Sonrasında nabızsız ventriküler taşikardi (VT) gelişti ve kardiopulmoner resüsitasyon ve 360 joul ile defibrilasyon yapıldı. 1 mEq/kg dan $NaHCO_3$ her 4 saatte bir verildi, bu tedaviden sonra QRS süresinin kısaldığı hastanın spontan solumaya başladığı görüldü. Propafenon zehirlenmesinde erken tanı ve uygun resüsitasyon girişimlerinin yapılması hayat kurtarıcı olabilmektedir. Hipotansiyon ve EKG bulguları varlığında $NaHCO_3$ tedavisi verilmesi gerekmektedir.

***Anahtar sözcükler**: İntoksikasyon; maling aritmi; propofenon.*

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Correspondence *(İletişim):* Dr. Ethem Acar. Erzurum Bölge Eğitim ve Araştırma Hastanesi, Acil Tıp Kliniği, 25040 Erzurum, Turkey
e-mail *(e-posta):* dr.ethemacar@hotmail.com

Introduction

Propafenone is a group 1C antiarrhytmic agent.[1] It is an agent used in patients with symptomatic supraventricular tachycardia requiring treatment such as atrioventricular (AV) node tachycardia and paroxysmal atrial fibrillation. It is also used in life-threatining symptomatic ventricular tachycardia requiring treatment. With excessive intake propafenone has serious side effects on cardiovascular, gastrointestinal, nervous, haematological and dermatological systems.[2,3] In propafenone intoxication, wide complex tachycardias, right bundle branch blocks, first degree AV block, prolonged QT interval, and generalized seizures can be observed.[3,4] Even though propafenone is a widely used class 1C antiarrhytmic, cases of fatal acute propafenone intake are rarely reported in the literature.[1]

In this report, we present a case of excess propafenone hydrocloride (HCl) intake with suicidal purpose and we aim to share our experience with malign arrhytmia development and arrhytmia management.

Case Report

A 22-year-old female patient presented to our emergency department with complaints of general situation distortion and generalized discomfort after ingesting 20 pills (6 g) of her friend's Propafenone HCl 300 mg for suicidal purpose one hour previously. On admission to emergency service, she had a general situation distortion with a consciousness level of stupor. She also had a superficial ventilation of 12/min, pulse of 64/min and blood pressure of 80/60 mmHg. Other physical examination findings were normal. The patient was transferred to the resuscitation room, supplemental oxygen therapy was initiated, an intrvenous (IV) route was obtained and endotracheal intubation was performed because of patient's inability to maintain an open airway. Blood glucose level checked from the finger tip was normal. The patient was given 1000 cc physiological saline in 15 minutes. In the electrocardiography (ECG), regular rhythm, wide QRS and the absence of p- wave was observed. We identified this as accelerating idioventricular rhythm. QRS duration was 148 ms and heart rate (HR) was 80 beats per minute (Fig. 1). Then 1 mEq/kg $NaHCO_3$ was administered by IV. A cardiology consultation was performed. The patient developed a generalized tonic-clonic epileptic seizure in the following one or two minutes which was ceased with 5 mg IV diazepam. Pulseless ventricular tachycardia developed and defibrillation with 360 joule was performed followed by cardiopulmonary resuscitation (CPR) (Fig. 2). After a 30 minute period of CPR in which pulseless VT develpoed five times and defibrillation with 360 joules was applied, pulsed rhytm was obtained. Heart rate was 90/min and blood pressure

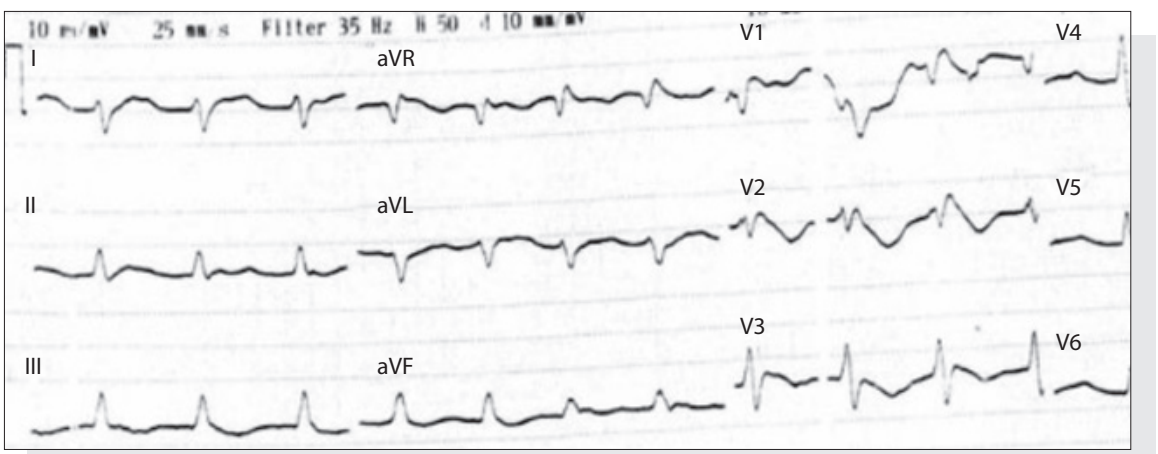

Figure 1. Accelerate idioventricular rhythm.

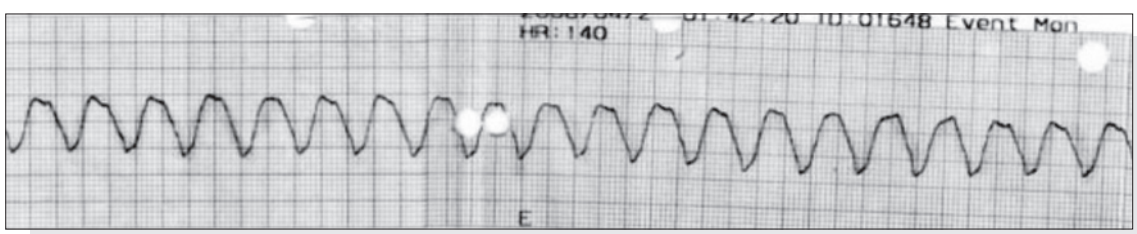

Figure 2. Ventricular tachycardia.

was 110/60 mmHg. In ECG, a sinus rhytm with a rate of 90/min was identified and the QRS duration was 134 ms. Laboratory findings were otherwise normal. $NaHCO_3$ administration of 1 mEq/kg every 4 hours was continued. After therapy, QRS duration shortened. The patient became conscious with spontaneous ventilation. She was then extubated and transferred to the coronoary intensive care unit. After a 3 day follow up in the coronary intensive care unit without additional problems, the patient was discharged with a total recovery.

Discussion

Propafenone may cause cardiac arrest through its proarrhytmic effects, either by causing new arrhytmias or worsening the present arrhytmia. These proarrhytmic effects may also be observed as bradicardias, conduction abnormalities (sinoatrial, atrioventricular or intraventricular blocks) or tachycardia (ventricular tachycardia). Rarely, ventricular flutter and/or fibrillation can be observed.[5,6] Thus, in this case malign arrhytmias were determined first as an accelerating idioventricular rhythm then as pulseless VT. It has been previously reported that intoxication with Group 1C antiarrhytmic agents has a higher mortality compared to many other types of intoxication.[7] In a study of 120 patients, Köppel et al.[8] reported that 34 of these patients were intoxicated with propafenone. Cardiac arrest developed in 29 of these patients and 27 of them did not respond to CPR. Stancak et al.[9] reported a 28 year old male with 4500 mg propafenone ingestion with suicidal intention. They reported that cardiopulmonary arrest developed in this case and no response to CPR was achieved. In a case reported by Üstündağ et al.,[7] a female patient with 7500 mg propafenone ingestion was presented. In this case cardiopulmonary arrest developed in 2 hours after admission and the patient died after 45 minutes of resuscitation. Available data reveals that hemodynamically unstable patients who need resuscitation have higher mortality rates. Even in our case, cardiopulmonary arrest developed. The patient responded to our early, effective and long lasting CPR.

Seizures may be seen with propafenon overdose, especially in high dose ingestions. Rambourg-Schepens et al.[4] reported a case with generalized seizure after 1 hour following propafenone intoxication. Our case presented with a depressed consciousness and developed an epileptic seizure.

Early monitoring, early diagnosis and early treatment was important to this patient's survival. Brubacher reported[10] that HCO_3 is considered by most toxicologists to be the treatment of choice for cardiac toxicity in the setting of sodium-channel blocker poisoning. It is reasonable to suspect that HCO_3 would benefit patients with propafenone toxicity, but the literature in this setting is limited. Ovaska et al.[11] reported a patient that was was managed with intravenous glucagon, sodium bicarbonate, hypertonic saline and this treatment was associated with a good outcome. Recently D'Orazio[12] reported that sodium bicarbonate bolus and infusion shortened the QRS length to 90 ms. To avoid persistent hypotension and ECG abnormalities, 1 mEq/kg sodium bicarbonate administration in every 4-6 hours is recommended.[13,14] In another study, Yi et al reported that Insulin treatment improved survival and delayed the hemodynamic and electrocardiographic consequences of propafenone toxicity. They reported that insulin prevented the decline of mean arterial pressure and heart rate. Insulin also prevented the increase of PR interval and QRS duration.[15] In our case, $NaHCO_3$ in an appropriate dosage was initiated IV for hypotension and QRS widening. $NaHCO_3$ therapy was maintained to retain ph of 7.50-7.55 until hypotension and QRS widening resolved.

When seizures occur, intervention with diazepam is recommended. In our case generalized tonic clonic type epileptic seizure was observed and treated with 5 mg diazepam. Additional seizures were not observed.

Efficacy of external or internal pacemaker implementation in propafenone intoxication is controversial because it is known that electrical pacing threshold increases due to this drug.[16] There are also reports that recommend hemodyalisis for treatment.[17] Our case improved both electrocardiographically and clinically after resuscitation, so cardiac pacing and hemodyalisis were not considered and the patient was evaluated by clinical follow-up.

Conclusion

Sodium bicarbonate administration in the presence of hypotension and QRS widening are vital. 1 mEq/kg sodium bicarbonate administration every 4-6 hours is recommended until hypotension and QRS widening rsolve. Early diagnosis, aggressive treatment, meticulous monitoring and supportive care can be vital in propafenone intoxication.

Conflict of Interest

The authors declare that there is no potential conflicts of interest.

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