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Successful treatment of suicidal mega dose of propafenone intoxication — a case report

Skuteczne leczenie przedawkowania propafenonu przyjętego w celu samobójczym

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INTRODUCTION

Propafenone is a Class 1C antiarrhythmic drug which also exhibits β-adrenergic and calcium channel-blocking activities. Intoxication with propafenone, in amounts greater than 4 g, is usually fatal, due to severe myocardial depression, refractory seizures, and ventricular dysrhythmias [1]. There are few case reports in the literature about intoxication with more than 4 g of propafenone [2–6]. We report here a case that was treated successfully after intoxication with 6 g of propafenone.

CASE REPORT

A 16 year-old girl was admitted to our emergency department (ED) having ingested propafenone (20 tablets, each containing 300 mg) in a suicide attempt. She was brought to the ED by ambulance one hour after ingesting the pills. Her physical examination revealed that she had altered mental status, with the Glasgow Coma Scale of 8. Her heart rate was 75 bpm, arterial blood pressure was 70/40 mm Hg, and capillary refilling time was 4 s at admission. She had respiratory distress and her pulse oxygen saturation was 78% with face oxygen mask. She was immediately intubated for airway protection. Her clinical condition rapidly deteriorated after the intubation and she developed cardiac arrest. Epinephrine (1 mg) and atropine (1 mg) were administered intravenously. Cardiopulmonary resuscitation was started and her heart rhythm became ventricular tachycardia with weak pulse. She received three electrical cardioversion shocks and the monitor demonstrated prolongation of QRS and QT intervals and electrical atrioventricular dissociation (Figs. 1, 2). Overall, the cardiac arrest was aborted within 3 min. She remained haemodynamically very unstable and received three intravenous normal saline bolus (20 mL/kg). Amiodarone (5 mg/kg) and lidocaine (1 mg/kg) were administered. In addition, she received dopamine

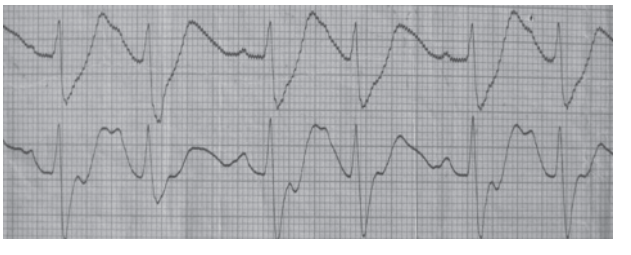

Figure 1. Ventricular tachycardia with weak pulse following cardiopulmonary resuscitation

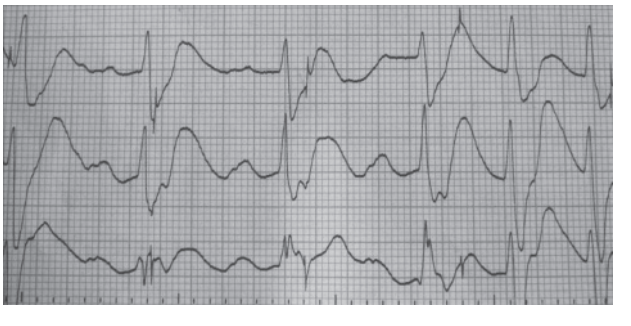

Figure 2. Complete atrioventricular block following cardiopulmonary resuscitation

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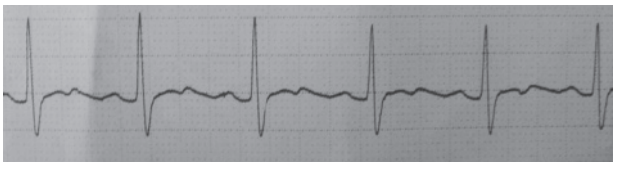

Figure 3. 34 hours after admission, her heart rhythm was normal. She had an temporary pacemaker and her corrected QT interval was prolonged (480 ms)

(10 μg/kg/min), dobutamine (15 μg/kg/min), norepinephrine (0.4 μg/kg/min), and epinephrine (0.3 μg/kg/min) to improve her persistent hypotension and haemodynamic instability. Her heart rate was severely bradycardic and a temporary pacemaker was placed through her right subclavian vein (Fig. 3). Unfortunately, her blood propafenone level could not be measured in our hospital.

She developed generalised tonic clonic seizures immediately after cardiopulmonary resuscitation. Her seizure was treated with intravenous diazepam followed by midazolam infusion. Gastric lavage was performed, and she received activated charcoal when she was haemodynamically stable. The patient was transferred to the paediatric intensive care unit where she remained haemodynamically unstable until her clinical condition started to improve by the end of the second day. At that time, the inotropic agents and sedatives were stopped, the temporary pacemaker was discontinued, and she was finally extubated. She was diagnosed with major depression and transferred to an inpatient psychiatric unit on the fourth day. She is now attending psychiatric outpatient follow-up visits.

DISCUSSION

Propafenone is an antiarrhythmic drug which also exhibits β-adrenergic and calcium channel-blocking activities. It is currently available in Turkey as 150 or 300 mg tablets. Propafenone can cause many ECG changes, particularly prolongation of the PR interval [1, 7–9]. Other known ECG changes include bundle branch block, widened QRS and QT intervals, ventricular tachycardia and bradycardia. Propafenone overdose may lead to congestive heart failure and hypotension. Seizure is an important clinical sign of propafenone intoxication. Why seizure should be associated with propafenone overdose remains unclear, but could be related to a specific toxic effect or cerebral hypoperfusion secondary to dysrhythmia and conduction disturbance induced by propafenone.

There are few case reports of propafenone intoxication with doses of 4 g to 9 g. No survived paediatric case has been described so far [1–5]. Our patient ingested 6 g of propafenone and presented with severe myocardial depression and life-threatening ventricular dysrhythmia. Her markedly abnormal ECG upon presentation is characteristic of acute propafenone toxicity, with profound slowing of cardiac conduction leading to widening of QRS complex, QT interval and prolongation of PR segment.

Propafenone intoxication is uncommon in children and young people. On the other hand, it is a frequently used drug in adults with cardiac dysrhythmias, a fact which makes propafenone an accessible drug. Propafenone overdose cases are relatively common among adults. In summary, propafenone should be kept in mind as a possible offending agent in children who present to the ED with cardiac failure, conduction disturbance, and seizures, especially following a suicide attempt.

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