Brugada Pattern, Brugada Phenocopy, What to Think?
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Introduction

Propafenone is a class I anti-arrhythmic medication with beta-adrenergic and calcium channel blocker properties.

Brugada syndrome (BrS) has a typical electrocardiographic pattern characterized by increased propensity for malignant ventricular arrhythmias and sudden death in patients with no structural heart disease.1

Brugada phenocopies (BrP) have electrocardiographic patterns that are identical to true type 1 and type 2 Br, despite the absence of a true congenital BrS. BrP are elicited by clinical conditions including ionic (or water and electrolyte) disturbances, myocardial ischemia and pulmonary embolism.2-5

We report a case of suicide attempt by an overdose of propafenone which yielded a type 1 Brugada pattern.

Case report

Seventeen-year-old adolescent admitted to the emergency department for voluntary intake of 40 tablets of propafenone 150 mg. The patient was hemodynamically stable at admission and conscious. There was no known family history of cardiovascular diseases, syncope or arrhythmias. Gastric lavage followed by activated charcoal (50 g) was performed, with emptying of food, but apparently not of medication. Ten minutes after admission, the patient had a generalized tonic clonic-seizure (with intravenous administration of midazolam 5 mg), followed by severe bradycardia that progressed to cardiac and respiratory arrest. Advanced life support (ALS) measures were started with return of spontaneous circulation (ROSC) after the fourth cycle. Electrocardiography (ECG) was performed and revealed a wide QRS and ST segment elevation in V1-V2 leads, consistent with a BS pattern (Figure 1). Arterial-blood gas test showed: pH 7.08; PCO₂: 51 mmHg; pO₂ 45 mmHg; Na 145 mmol/L; K 3.6 mmol/L; Cl 113 mmol/L; Lact 9.4 mmol/L; HCO₃ 14.8 mmol/L. A second cardiorespiratory arrest occurred 20 minutes after ROSC, followed by three cycles of ALS and ROSC. For hemodynamic and rhythm stabilization, a temporary pacemaker was implanted by right femoral artery access. Norepinephrine at 0.1 mcg/kg/min and sodium bicarbonate 8.4% were administered for recovery of pulse rate. Complete reversal of the signs of toxicity was observed three hours after hospital admission. ECG then revealed sinus rhythm with no BrP features (Figure 2).

Discussion

BS is a rare, genetically determined condition with autosomal dominant transmission.1

Today, the diagnosis of BS is defined by the presence of type 1 ECG in at least two right precordial leads, combined with one of the following: documented ventricular fibrillation (VF) or ventricular tachycardia (VT), family history of cardiac sudden death (< 45 years), type 1 ECG in other members of the family, VF/VT in programmed electrical stimulation or syncope.6

Diagnosis may be difficult in cases of borderline or Brugada-like repolarization patterns with or without symptoms.5

Recent studies have reported cases of type 1 Brugada pattern at ECG caused by underlying causes, with normalization of ECG. Theses cases were classified as type 1 Brugada pattern.2-5 BrP have been classified by

Keywords

Brugada syndrome / genetic; Propafenone; Anti-Arrhythmia Agents; Suicide; Death, Sudden, Cardiac; Arrhythmias, Cardiac; Electrocardiography.
their etiologies, that include metabolic conditions such as hyperkalemia, mechanical compression, myocardial ischemia, pulmonary embolism, myocardial disease, pericardial disease and following catheter ablation.6

Electrocardiographic changes of BrP are identical to those of type 1 and type 2 Brugada pattern, with some different characteristics:2-5

1. Identifiable underlying condition;
2. Resolution of the ECGC pattern once the underlying condition has been treated;
3. Low pretest probability for BrS established by lack of symptoms and no family history of BrS;
4. Negative provocative testing with a sodium channel blocking agent;
5. Negative genetic testing

Propafenone has many known side effects, including increase of PR interval, widening of the QRS complex, bundle branch block, ventricular arrhythmias, bradycardia and hypotension. However, there are few reports on the effects of an overdose of propafenone, which have speculated the mechanisms of calcium channel inactivation even in the absence of a genetic mutation.2-11

“BrP” do not include type 1 Brugada pattern induced by calcium channel blockers and hence we could not find a correct classification of our case reported.

If on one hand some authors suggest provocative testing for patients with a “non-type 1” Brugada pattern to provide lifestyle counseling on fever management and use of provocative drugs, on the other hand, some advocate that there is no gain in performing this test in asymptomatic, “non-type 1” Brugada pattern patients for risk stratification. Thus, the role of provocative tests in patients with type 1 Brugada pattern in intoxication by propafenone has not been established.

In the case reported, we decided not to perform the provocative test; the adolescent is well, under no psychiatric treatment.9

Conclusion

Propafenone intoxication is a rare event and, to our knowledge, there is no detailed epidemiologic study on this in the literature.5,11

Despite the severity of the condition, successful stabilization of the individual was achieved by early resuscitation, invasive mechanical ventilation, transitory stimulation, and correct management of acidosis. This case emphasizes the importance of distinguishing the toxicological effect of BS from that of BrP.

Author contributions

Conception and design of the research: Martins JL, Ferreira R. Acquisition of data: Martins JL. Analysis and interpretation of the data: Martins JL. Writing of the manuscript: Martins JL, Ferreira R. Critical revision of the manuscript for intellectual content: Martins JL, Ferreira R, Viana J, Santos J.

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References