Research Article

Acute Voluntary Poisoning with Acebutolol Managed in the Emergency Department

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Received Date: November 14, 2020

Publication Date: December 01, 2020

Abstract

A case of nonfatal acebutolol intoxication in an 18-year-old female is described. Ingestion of 8000 mg (20 tablets) Acebutolol resulted in a cardiogenic and vasoplegic shock and membrane-stabilizing effect 3 hours after ingestion. The patient remained conscious, had severe hypotension and the electrocardiogram showed, as in the poisonings with sodium channel blockers, a prolonged QRS duration of about 140 milliseconds. After the Half-Molar Sodium Bicarbonate neutralization of the plasma and the administration of 3 mg of Glucagon membrane-stabilizing effect has disappeared and the blood pressure gradually increased to normal values in 2 hours time.

Keywords: *Acebutolol, Poisoning, Sodium Bicarbonate, Glucagon, Emergency department.*

Introduction

Acebutolol is a β1-selective adrenergic receptor antagonist with moderate membrane-stabilizing activity and intrinsic sympathomimetic activity; therefore, the drug is indicated in hypertension, myocardial infarction and arrhythmia **(**1**)**. However, acebutolol and other agents with membrane-depressant effects further depress myocardial contractility and conduction and may be associated with QRS intervals abnormalities and predispose the patient to ventricular tachydysrhythmia. Even though acebutolol is commonly prescribed, few cases of fatal poisoning have been reported to date **(**2–7**)**. In the β Blockers poisoning the precocity of the antidote treatment and emergency resuscitation procedures determine the prognosis. Here, we report a nonfatal case of acebutolol self-poisoning in an 18-year-old woman.

Patient and Observation

Case Presentation

An 18-year-old woman, with a depression history, who presented in the emergency department, accompanied by her mother, because of agitation following a family conflict. We found the notion of 4 suicide attempts by voluntary multi-drug poisoning. On arrival the patient was aggressive with a refusal to contact the medical team, according to the mother's questioning, it was a usual reaction following a family conflict and that there were no suicidal intentions.

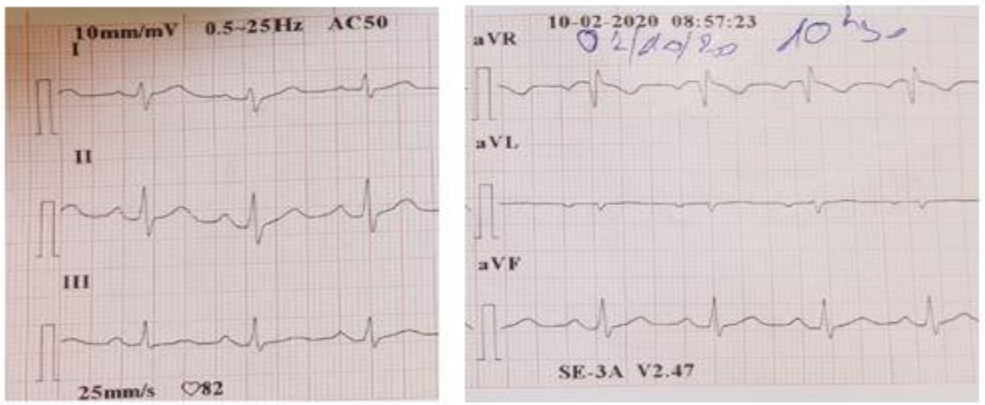

Figure 1 (a, b): ECG on admission in the Emergency Department, normal QRS duration

On admission, physical examination, as well as ECG were normal (Figure 1, “a” and “b”). After two hours the patient calmed down and accepted an interview with the psychologist of the emergency department who noticed that she was more asthenic than on arrival. During the psychotherapy session, the patient declared to having ingested two boxes of her father's treatment 3 hours ago. At the resumption of the medical interrogation, she would have voluntarily ingested 20 tablets of Acebutolol (Sectral®) at 400 mg, i.e. a cumulative dose of 8000 mg. On immediate admission in the emergency room, his blood pressure was 80/40 mmHg, pulse and respiratory rates were 72/minute and 22/minute, respectively. His body temperature was 37.2°C and she was conscious. The auscultation showed rapid pulse and no pulmonary rales. In the ECG, the heart rate was 75/minute with QRS prolongation of about 140 milliseconds (Figure 2, “a” and “b”).

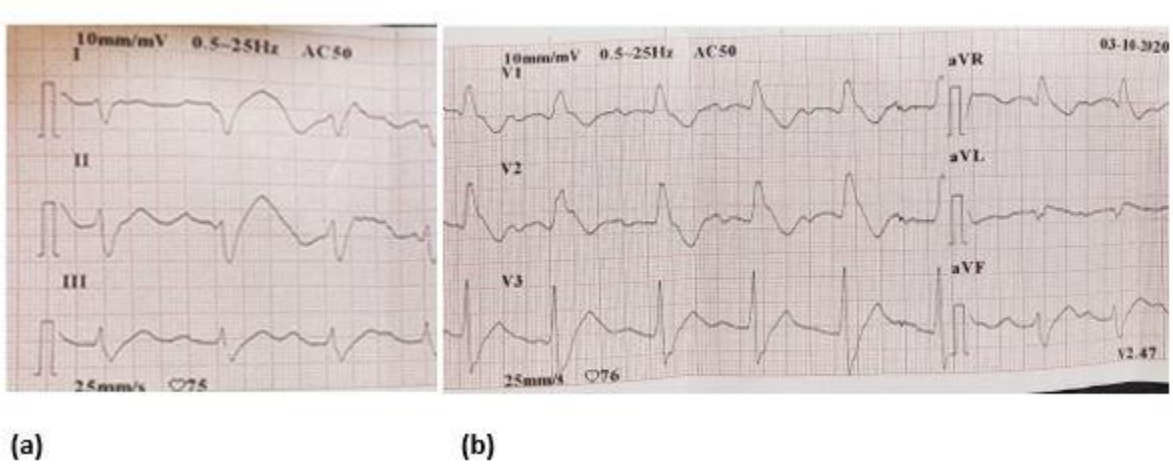

Figure 2 (a, b): ECG after 2 hours, wide QRS > 140ms

Investigations

Blood tests were normal (hemoglobin 12 g/dl, thrombocytes 250×109/L, leucocytes 8.2×109/L) negative C reactive protein. There was no coagulopathy (D-dimer, prothrombin time and partial thromboplastin time values were normal) no ionic disorders and the serum glucose levels were normal. Blood gases showed hyperlactatemia (2.86 mmol/L) with a corresponding acidosis (pH= 7.34).

Treatment and Monitoring

Epinephrine has been used to resuscitate the cardiogenic and vasoplegic shock. Half-Molar Sodium Bicarbonate (42 O/OO) was administrated to treat sodium channel blockade (500 mL) with an initial dose of glucagon (3 mg). Fluid resuscitation was about 500 ml. The patient was kept under continuous cardiac monitoring and a 12-lead electrocardiogram in the emergency room to control cardiac conduction abnormalities.

Outcome

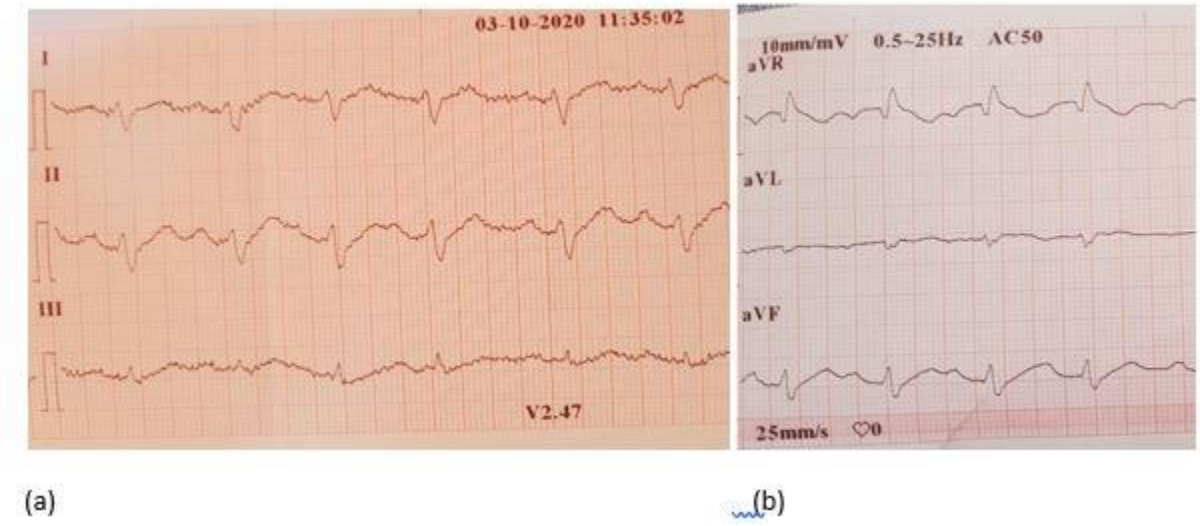

Figure 3 (a, b): ECG after initial resuscitation in the emergency room, the duration of the QRS decreased to 110-120 ms.

After one hour the duration of the QRS decreased to 110 ms (Figure 3, “a” and “b”), hemodynamic state was stabilized with decreasing doses of adrenaline. The patient was transferred to the intensive care unit, the evolution was favorable she was discharged after 48 hours and referred for a psychiatric consultation.

Discussion

B-adrenergic blockers (BB) remain a common treatment for various cardiovascular and other medical conditions **(**8**)**. Unfortunately, they also remain frequent causes of cardiovascular collapse following accidental or intentional overdose. We report a case of nonfatal acebutolol intoxication in an 18-year-old female that resulted in a cardiogenic and vasoplegic shock and membrane-stabilizing effect 3 hours after ingestion. Managed with fluids and catecholamines support Half-Molar Sodium Bicarbonate neutralization and administration of Glucagon.

Adrenergic receptor agonists are a rational therapeutic choice in drug-induced shock for their cardiotonic and vasoactive effects. All of the available catecholamines, including dopamine, dobutamine, epinephrine, and norepinephrine, have been used to resuscitate BB **(**9,10**)**. In general, there is no single agent that is predictably successful for all cases. In theory, the choice of adrenergic agonist could be based upon the pharmacologic activity of the offending agent. For example, the patient who has depressed contractility and decreased peripheral resistance may benefit from epinephrine, because this drug possesses both b- and a-agonist properties.

Sodium bicarbonate therapy is used to treat sodium channel blockade and academia. BB drugs appear to antagonize myocardial sodium channels and induce the so-called "membrane-stabilizing effect" include acebutolol, betaxolol, carvedilol, metoprolol, oxprenolol, and propranolol beta-Adrenergic blocking drugs have been available for several years to treat ischemic heart disease and other cardiovascular and noncardiovascular disorders. There are multiple drugs in this class with various pharmacodynamic and pharmacokinetic properties that may be important in specific clinical conditions. Thus, toxicity from these drugs may include widened QRS in addition to bradycardia **(**11**)**. Sodium bicarbonate is the traditional treatment for wide complex QRS conduction abnormalities due to sodium channel antagonism. It may be a useful adjunct to other resuscitation measures in cases of BB toxicity with QRS prolongation greater than 120 milliseconds.

Antidotal glucagon use in BB toxicity started modestly, then increased and many subsequent reports described good clinical response often after conventional therapy failed **(**12**)**. The recommended initial dose of glucagon is 50 to 150mg/kg, roughly 3 to 10 mg in a 70-kg patient. Smaller initial doses frequently fail to produce adequate cardio dynamic responses **(**13,14**)**. Glucagon works rapidly. Responses in heart rate and blood pressure often occur within minutes **(**15**)**. Bolus therapy may be repeated in 3 to 5 minutes.

Many nonpharmacologic modalities were used to resuscitate BB Toxicity, like Hemodialysis. Extracorporeal drug removal has limited use fullness following BB overdose. Extracorporeal circulatory support, aortic balloon pump, and prolonged cardiopulmonary resuscitation (CPR) have been employed in severe toxicity when standard pharmacologic measures failed **(**16**)**. Following a massive propranolol overdose that resulted in a witnessed cardiac arrest and 4 hours of CPR, 6 hours of extracorporeal support resulted in full neurologic recovery.

Continuous cardiac monitoring and 12-lead electrocardiogram are essential to identify cardiac conduction abnormalities. Because several BBs can antagonize myocardial fast sodium channel function similar to that of tricyclic antidepressants, the 12-lead electrocardiogram will also assess QRS duration and act as a treatment indicator.

The overall objective of the initial therapy is to improve organ perfusion with subsequent increases in survival. Reasonable clinical and physiologic markers of the efficacy of therapy include increased blood pressure (R90 mm Hg in adult); adequate heart rate (60 bpm); resolution of acidemia, euglycemia, adequate urine flow (1–2 mL/kg/hour) and reversal of cardiac conduction abnormalities (QRS <120 milliseconds.

Conclusion

We reported a case of oral acebutolol self-poisoning resuscitated rapidly and efficiently in the emergency room. The prognosis of self-poisoning with beta-blockers can be excellent if medical management is codified and started immediately.

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Volume 1 Issue 4 December 2020