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Atropine intoxication: A case presentation

Ahmet Yüksek; Elif Doğan Bakı*

Department of Anesthesiology and Reanimation, Afyon Kocatepe University, Turkey

***Corresponding Author(s): Elif Doğan Baki,**
Anesthesiology and Reanimation Department, Faculty of Medicine, Afyon Kocatepe University, Turkey
Email: elifbaki1973@mynet.com

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Abstract

Anticholinergic drug overdose is a difficult condition. Atropine or antimuscarinic drug overdose is usually an unwanted development during treatment. Symptoms vary widely from tachycardia to coma. It should be considered especially in unconscious patients.

The case is here reported of intentional atropine overdose. A 29-year old male pharmacy staff member self-administered 10mg intravenous atropine with the intention of suicide in the hospital. Within minutes the patient was brought to the Emergency Department. Heart rate was measured as 170/110 at 165 blood pressure. Body temperature was normal. After 10 minutes in the Emergency Department, the respiratory distress and agitation of the patient began to increase and the patient was intubated for airway control, then admitted to the ICU. This is one of the few examples in literature of the use of atropine for suicide purposes.

Introduction

Atropine is a natural antimuscarinic drug. It inhibits acetylcholine from acting on smooth muscle, the central nervous system and parasympathetic areas of secretory glands. It decreases secretions and increases cardiac output. This effect is used in the treatment of symptomatic bradycardia. Atropine overdose is usually an unwanted development during treatment. Indications of toxicity can be seen with 5-10 mg of atropine. These findings are widespread and can range from tachycardia to coma. The aim of this case report was to share our Emergency Department and intensive care approach to atropine overdose in the hospital. There are very few reports in literature about the use of high-dose atropine for suicide.

Case Presentation

A 29-year old male pharmacy staff member self-administered 10mg intravenous atropine in the hospital with the intention of suicide. Within minutes the patient was brought to the Emergency Department (ED). In the first examination findings in ED, the patient was conscious, nervous and tachycardic. The patient had exophthalmus and impaired accommodation. He was monitored, and blood pressure was measured as 170/110 mmHg with a heart rate of 165 beat/minute. Body temperature was normal. After 10 minutes in ED, the respiratory distress and agitation of the patient began to increase and the patient was intubated for airway control and transfered to the İntensive Care Unit (ICU). In the ICU, ECG was again applied. We have not observed any other ECG findings other than sinus tachycardia. Heart rate was monitored at 145 bpm. No additional cardiac drug was administered because the sinus rhythm was present. Midazolam infusion was started. Physiostigmine was not available in the hospital, so could not be given. As there were determined to be peripheral effects, 0.5mg neostigmine was administered and a midazolam infusion started. Tachycardia was strained at the end of the second hour and at the 4th hour, the midazolam infusion was halted. Consciousness recovered and the patient was extubated. After counselling by the Psychiatry Dept, the patient was discharged on the 3rd day.

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Discussion

Atropine, a natural belladonna alkaloid, antagonizes acetylcholine at the neuromuscular junction and is a competitive antagonist for cholinergic receptors. However, only muscarinic receptors are blocked. Cardiac muscle, exocrine glands, and smooth muscle are the most affected areas. The most difficult blocking receptor is gastric acid secretion and pancreatic exocrine secretion [1]. The use of atropine for suicide is not common. In general, anticholinergic syndrome can be seen as a side-effect in drug mismatches, in older patients due to multiple drug use, or as a side-effect of herbal medicines. In 2008, a study was conducted with anticholinergic drugs and plants at the American Association of Poison Control Centers (AAPCC). In that study, 20,000 patients were examined and no mortal cases were observed. The number of patients with anticholinergic side-effects in a year is not very low [2].

There have been few cases reported in literature of atropine use for suicide. At an autopsy in Germany, atropine intoxication was observed and in the postmortem toxicology examination, a high dose of atropine was determined in the blood, urine and hair samples. That case is an interesting example of the use of atropine eye drops for suicide [3].

Anticholinergic syndrome should be considered in all patients admitted to ED with unexplained consciousness discomfort because intoxication with anticholinergic effects also occurs with drugs other than atropine [4]. The differential diagnosis of anticholinergic toxicity includes life-threatening tables such as viral encephalitis, Reye's syndrome, head trauma, alcohol and sedative hypnotic withdrawal syndrome, postictal status, other intoxications, neuroleptic malignant syndrome and acute psychotic disorder [5].

Atropine is metabolized mainly in the liver, and half life is 2.5 hours. Normally, the maximum dose is not precisely defined for the use of atropine. Side-effects begin to develop at doses of 5-10 mg. The most common findings due to excessive doses are tachycardia, dyspnea, fever, central nervous system findings, and dryness in the mucous membranes. These side-effects can be seen in a wide range from gastric late emptying to confusion, delirium and coma. Dose response may vary according to the patient [1,6]. In a case report published in Germany, the patient was mistakenly administered 20mg atropine and no serious central nervous system or respiratory signs were observed. However, peripheral effects such as redness on the skin, drying of mucous membranes and fever have been observed at high doses. The dose used in that reported case was twice that taken by the current patient and the different responses to two different doses indicate that atropine dose response is variable [7].

Early treatment of atropine overdose can be life-saving. Stabilisation of cardiac circulation and neurology should be attempted in prehospital care. Patients with central nervous system findings or with respiratory insufficiency should be intubated. The current patient was intubated for airway control at the onset of respiratory distress. Pre-hospital physiostigmine is not recommended. Benzodiazepine treatment for seizures can be used. The most common ECG finding in these patients is sinus tachycardia and stability does not require treatment in patients. The right axis or the QRS extension (>100 milliseconds) can be monitored and in these cases, sodium bicarbonate can be given. Anticholinergic drugs may cause ECG changes such as progressive prolongation of PR, QRS, and QT intervals in humans and animals by affecting Ca++, Na+ and especially K+ channels. If it exists, sodium bicarbonate, lidocaine, magnesium or overdrive pacing may be given for treatment. The current patient had sinus tachycardia at a heart rate of 140 bpm and began to regress at the end of the second hour. We did not observe any prolonged PR, QRS, and QT intervals at ECG. No additional therapy was needed for cardiac stabilization.

Most anticholinergic drugs have a high distribution volume and are highly bound to the protein. Hemodialysis or hemofiltration may not work for this reason [5].

Anticholinesterase drugs are used as overdose treatment of anticholinergics. The most commonly used anticholinesterase drug is physostigmine, which is a quaternary ammonium compound that inhibits AchE by crossing the blood brain barrier. The central and peripheral effects increase the amount of acetylcholine at the neuromuscular junction, allowing parasympathetic conduction [5]. In a retrospective study comparing physostigmine and benzodiazepines in anticholinergic poisoning treatment, it was reported that benzodiazepines reduced delirium in 24% of patients and physostigmine reduced delirium in 87% of patients and controlled agitation in 96% of patients. However, physostigmine is not a drug that can be easily found in Turkey. There was no physostigmine in our pharmacy that day and there was not enough time to obtain physostigmine because the findings were rapidly developing following the use of high-dose atropine. Neostigmine can be used for peripheral findings, although the effect is less than that of physiostigmine [8,9].

Anticholinergic poisoning may be discounted if patients with suspected toxicity do not have evidence of toxicity and if anticholinergic symptoms do not develop after 6 hours of follow-up. Many symptomatic patients usually require at least 24 hours of follow-up. It must not be forgotten that the toxicity effects may be anticipated and recurrent toxicity may occur, as the half-life of physostigmine is shorter than the half-life of some anticholinergic agents. Patients receiving physostigmine have to stay for longer periods of observation. Although physostigmine could not be used in the current case, a follow-up period was required for some time after the symptoms of the patient had returned. The patient was discharged on the 3rd day after being observed for 2 days.

Conclusion

Anticholinergic drug overdose is a difficult condition. Symptoms can vary widely, ranging from tachycardia to coma. It can be observed with many medicines besides atropine and should be kept in mind especially in elderly patients who are using a combination of drugs, and particularly when the patient is unconscious. Specific treatment is physostigmine and as symptoms can develop rapidly, it is vital that this medicine is kept in hospital pharmacy stock.

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