Case Report

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A Case Report of Atropine Psychosis in Organophosphorus Poisoning: Glycopyrrolate used Successfully as a Replacement

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Abstract

Atropine is the most commonlyused drug for the treatment of organophosphorus poisoning which may occasionally lead to central nervous system side effects like psychosis and delirium. Discontinuation of atropine, modification of the dosage or replacement with other medications may be required alongwith antipsychotics and sedatives in such cases. We report herein a case of suicidal ingestion of organophosphorus insecticide (ANACONDA) who received atropine infusion for the management of muscarinic symptomsbut developed psychotic features during treatment. Atropine was discontinued and replacement done with glycopyrrolate infusionwhich could successfully counteract the muscarinic features. The use of glycopyrrolate as replacement in atropine psychosis and the recommended dosage regimen has been sparsely described in literature.

Keywords: Organophosphorus poisoning; Atropine psychosis; Glycopyrrolate; Bronchorrhoea**.**

Introduction

Atropine is a competitive, reversible antagonist of muscarinic acetylcholine receptors which is most commonly found in Atropabelladona, the deadly nightshed and some datura species. Due to its parasympatholytic activity, it is widely used in the treatment of bradycardia of different aetiologies, some types of conduction blockade, poisoning by organophosphorus insecticides and nerve gas, reducing bronchial secretion and as topical ophthalmic solution for therapeutic mydriasis and cycloplegia. Atropine may cause photophobia, dryness of mouth, warm and dry skin, decreased lacrimation, tachycardia and arrhythmia as side effects. Psychotic symptoms such as delirium, hallucinations, confusion, agitation etc. may also occur because of the ability of atropine to cross blood brain barrier. Susceptibility to doses causing these toxic reactions may vary from individual to individual.[1] We herein describe a case of organophosphorus poisoning who developed atropine psychosis during the course of treatment which improved on discontinuation of atropine and adding glycopyrrolate as replacement for improving the muscarinic symptoms.

Case Report

A 33 years old man presented to emergency department with alleged history of insecticide ingestion (Anaconda) which contains organophosphorus compounds, chlorpyrifos and cypermethrine, followed by multiple episodes of vomiting. On examination, patient was conscious and oriented with a pulse rate of 64/min, blood pressure of 120/70 mmHg, respiratory rate of 22/min, oxygen saturation of 94% in room air, with excessive sweating and salivation. On auscultation mild crepitations were detected over bilateral lung fields. Both the pupils were constricted and sluggishly reacting to light. Gastric lavage was done using normal saline. Injection atropine 1.2 mg was administered intravenously and 2.4 mg repeated after 10 minutes and injection pralidoxime 2 gm was administered as stat dose intravenously before shifting to intensive care unit. Fluid resuscitation was done using ringer's lactate and normal saline and urine output was maintained at around 50-100 ml/hour. Patient continued to have excessive respiratory secretions and a heart rate of 55-70/min. Atropine infusion was started @ 15 ml/hour (0.06 mg/ml) along with injection pralidoxime 1 gm intravenously twice daily, injection ranitidine 50 mg intravenously twice daily, injection thiamine 100 mg intravenously once daily and salbutamol nebulization. Initial arterial blood gas analysis, complete blood count, renal function test, liver function test, coagulation profile, serum electrolytes and chest x-ray were all unremarkable. Heart rate, blood pressure, SpO_2, ECG, respiratory rate, urine output and pupil size were continuously monitored. After 6 hours of atropine infusion, patient started developing agitation, hallucination and confusion suggesting central anticholinergic toxicity. Infusion atropine was immediately stopped and intravenous injections of haloperidol 5mg and midazolam 1 mg were administered. Other causes or drugs with the potential to causethe central nervous system (CNS) symptoms were ruled out. Patient continued to have bilateral chest crepitations, excessive salivation, sweating, mildly constricted pupils and heart rate that was dropping below 80/min. Injection glycopyrrolate 0.4 mgwas administered intravenously and infusion started @ 0.3 mg/hour as replacement for reversing the muscarinic symptoms and titrated with intense monitoring of vital parameters. Intermittent doses of injection haloperidol and midazolam were administered. Symptoms of atropine psychosis completely subsided within 24 hours of discontinuation of the drug. After complete disappearance of muscarinic symptoms glycopyrrolate infusion was gradually tapered and then stopped. Patient required glycopyrrolate infusion for a total of 36 hours.

Discussion

Organophosphorus compounds act by irreversibly binding and inhibiting the acetyl cholinesterase enzyme, resulting in excessive accumulation of acetylcholine producing SLUDGE symptoms (salivation, lacrimation, urination, diaphoresis, gastrointestinal upset, emesis) along with bradycardia, hypotension, shock, respiratory weakness and paralysis. Atropine is used to reverse these cholinergic symptoms to the targeted end point of decreased respiratory secretions and clear chest. Atropine however is unable to reverse muscular weakness due to lack of effectiveness at nicotinic receptors.[2] Pralidoxime when used within 48 hours of organophosphorus ingestion reactivates phosphorylated acetyl cholinesterase enzyme by binding to organophosphorus compound. Oximes may be useful upto 120 hours of ingestion of diethyl organophosphorus.[3] Atropine, unlike glycopyrrolate, has the potential for CNS penetration reversing the central effects like coma and depressed respiration and is thus the antimuscarinic agent of choicein organ ophosphorus poisoning. However, this property of atropine attributes to the symptoms of psychosis and delirium too. These CNS symptoms require prompt discontinuation of atropine, modification of the dosage or replacement with another drug like scopolamine, physostigmine or glycopyrrolate along with antipsychotic and sedatives.[4] In our case, atropine was stopped owing to the development of psychotic symptoms prior to complete atropinisation. Peripheral muscarinic effects like bronchorrhea and chest crepitations were still present which required initiation of glycopyrrolate therapy. A combination of atropine and glycopyrrolate used in organophosphorus poisoning by Regan et al. found a lower mortality rate without influencing the rate of atropine toxicity.[5] Peter et al. reported a case of organophosphorus poisoning with intermediate syndrome where glycopyrrolate was used to treat bronchorrhea that was resistant to atropine.[6] Glycopyrrolate being twice as potent as atropine the dose requirement to reverse the peripheral muscarinic symptoms is half of that of atropine.[7] In our patient lesser dosage of glycopyrrolate equivalent to one-third of that of atropine reversed the muscarinic effects completely.

Conclusion

The susceptibility and dose of atropine leading to undesirable CNS symptoms vary from individual to individual. Discontinuation of atropine followed by replacement with another anticholinergic with poor brain penetration like glycopyrrolate may be required to counteract the peripheral muscarinic effects. Titration of appropriate dosage of glycopyrrolate to achieve the desired effect is challenging and simultaneous intense monitoring is vital.

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