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International Journal of Anesthesiology & Research (IJAR)
ISSN 2332-2780

Organophosphate Intoxication Due to Suicidal Intent

Case Report

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Abstract

Organic phosphorus (OP) intoxication comprises a significant portion of intoxication due to agricultural chemicals. This type of intoxication occurs by oral intake, inhalation or skin contact accidentally during use or due to suicidal intent. It is most commonly observed in males from 15-45 years. OP reversibly inhibits the acetylcholinesterase (AChE) enzyme in the synapses. Symptoms and findings appear 1-2 hours after exposure generally linked to the balance between the nicotinic and muscarinic receptors. Treatment comprises general support treatment, decontamination, prevention of absorption and administration of antidote. In this study we present a case with organophosphate intake by the oral route with suicidal intent. We discuss the precautions necessary for OP intoxication and the clinical monitoring and treatment of this patient in intensive care.

Introduction

Organophosphates are one of the chemical materials (organophosphates, carbamates, organic chloride, pyrethrins) commonly used as insecticides in industry, agriculture and homes. Diazinon, ortenna, malathion, parathion and chlorpyrifos are the most commonly used chemicals in the organophosphate group. Acetyl choline is a neurotransmitter found in parasympathetic and sympathetic ganglions, postganglion parasympathetic nerve endings, neuromuscular junctions and sweat glands and is destroyed by acetylcholinesterase and butyrylcholinesterase. OPs bind to phosphate radicals with covalent bonds in the active region of the acetylcholinesterase enzyme found in the central nervous system and erythrocytes and butyrylcholinesterase enzyme in plasma, irreversibly inhibiting these enzymes [1]. The result is that nicotinic and muscarinic cholinergic receptors in the neuromuscular junctions of the central and autonomic nervous system are overstimulated by the effect of acetyl choline accumulating at cholinergic junctions and a clinical tableau occurs. The severity of the tableau varies depending on the amount and type of the agent, exposure route, rate of absorption, destruction rate and previous patient history of exposure to cholinesterase inhibitors. OPs are rapidly absorbed by gastrointestinal, lung, skin and mucous membranes and the conjunctiva. Clinical complaints generally occur 1-2 hours after exposure with nicotinic, muscarinic and central nervous system (CNS) findings and cardiopulmonary symptoms. Diagnosis is made based on history, related symptoms and reduced cholinesterase levels. Treatment is ensured by airway reliability, strong respiratory support, symptomatic support, decontamination, prevention of absorption and administration of antidote. In this article we describe the treatment and monitoring of a case developing clinical symptoms 1-2 hours after oral organophosphate intake with suicidal intent in intensive care.

Case

A 43-year old male patient weighing 70 kg was brought to the emergency services by ambulance due to sudden loss of consciousness. Pupils were myotic, there were increased oral secretions and the patient had wheezing respiration and complete loss of consciousness. The patient had an intense smell resembling garlic. Vital signs were blood pressure: 90/60 mmHg, pulse: 52/min, respiration rate: 20/min and fever 37 °C. The patient was intubated and airway support ensured. Based on clinical findings and symptoms, organophosphate intoxication was considered as leading diagnosis. The patient was a bus driver with no additional diseases, had no history with insecticides or agriculture so intake of chemical material with suicidal intent was considered a possibility. His stomach was washed with gastric lavage and 1 mg/kg active carbon was administered. The patient's clothes were

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Received: March 25, 2017
Accepted: April 20, 2017
Published: April 25, 2017

Citation: Kayir S, Dogan G, Ozek OC, Ekici AA, Asici E (2017) Organophosphate Intoxication Due to Suicidal Intent. *Int J Anesth Res.* 5(4), 439-441.
doi: http://dx.doi.org/10.19070/2332-2780-1700090

removed and he was washed with soap and water. At 3-5 minute intervals until secretions reduced he was administered a total of 20 mg atropine intravenously (IV). Laboratory investigations found glucose: 162 mg/dl, AST: 23 U/L, leukocytes 24500/uL and pseudocholinesterase: 101 U/L. The patient's ECG showed sinus bradycardia with rate of 52/min. PA found no pathological lung findings. The diagnosis of organophosphate intoxication was strengthened by pseudocholinesterase suppression and to prevent enzyme aging, 2 g pralidoxime (2-hydroxyminomethyl-1-methyl pyridinium chloride: 2-PAM) loading dose was administered. The patient was admitted to anesthesia intensive care for tests and treatment. GCS was assessed as 4. Mechanical ventilation was begun. For maintenance 100 mg/h 2-PAM infusion was begun. In the first 2 days in intensive care 1 mg/h iv atropine was administered. Prophylaxis treatment for pneumonia, pulmonary thromboembolism and peptic ulcer was added. On the 1st day after admission to intensive care he began parenteral feeding with enteral feeding begun on the 4th day. In spite of 2-PAM treatment, pseudocholinesterase levels were 101 U/L on the 1st day, 169 U/L on the 2nd day, 183 U/L on the 3rd day, 199 U/L on the 4th day, 208 U/L on the 5th day and 215 U/L on the 6th day. In spite of available treatments, the patient developed cardiac instability on the 6th day in intensive care. The hypotensive patient was administered 0.5-2 mcg/kg dopamine hydrochloride and 2.5-10 mcg/kg/min dobutamine hydrochloride. Despite support treatment, the patient remained hypotensive and was exitus on the 7th day.

Discussion

Organophosphates are compounds used to kill insects in many areas of life, like agriculture, animal husbandry, homes and workplaces. Organophosphate intoxication occurs at similar rates in nearly all countries in the world. Intoxication generally is seen due to accidents in homes, agriculture and industry (those working in production and transportation of this material) and in those working in the insect area. It is most commonly used with suicidal intent by males from 30-50 years of age. Due to the high incidence of occupational exposure, this intoxication is most common in males from 15-43 years of age. Intake by small children is generally accidental and the incidence of severe intoxication is higher [2-4]. Our case was a bus driver, with no activity related to agriculture or insecticides in the recent past according to his family. Due to these reasons, we considered that our 43-year old patient may have taken oral organophosphates with suicidal intent as a leading diagnosis. Systemic absorption of organophosphates occurs through inhalation, transdermal, transconjunctival and gastrointestinal routes. Depending on the organophosphate compound, intoxication may be severe or mild. Organophosphate compounds like methamidophos and methyl parathion are severely toxic, diazinon and dichlorvos are moderately toxic and malathion and bromophos are mildly toxic [6]. As our case ended with mortality, we believe he drank a large amount of organophosphate compounds like methamidophos or methyl parathion. Clinically organophosphate intoxication may cause muscarinic effects like myosis, bradycardia, bronchospasm, increased secretions (bronchal, saliva, tears, nasal, sweat), hypothermia and incontinence; nicotinic effects like mydriasis, tachycardia, hypertension, fasciculations, muscle cramps and muscle weakness; and CNS effects like depression, agitation, confusion, delirium, convulsions and coma [5]. If cholinesterase levels fall by 60%, headache and parasympathetic stimulation develops. Moderate symptoms like muscle weakness, tremor and neuropsychiatric symptoms occur with 60-90% reduction. With more than 90% reduction, severe symptoms of seizures, cyanosis, pulmonary edema, respiratory failure, coma and death may occur [7-9]. In our case there was more than 90% reduction in cholinesterase levels and as a result the muscarinic effects of bradycardia, myosis, increased secretions, bronchospasm, and respiratory failure and the central effects of syncope and coma developed. Diagnosis on suspicion of organophosphate intoxication is based on anamnesis, presence of significant toxicity and laboratory cholinesterase levels [7, 11]. Our patient's anamnesis suggested he may have attempted suicide. The smell emanating from the patient, clinical findings and most importantly suppressed pseudocholinesterase levels led to consideration of organophosphate intoxication. Treatment for organophosphate intoxication is decontamination, prevention of absorption, respiratory support and administration of antidote [6]. To ensure air way support for our case he was intubated and placed on a mechanical ventilator. The patient's whole body was washed with lots of water, gastric lavage was applied and active carbon administered. The specific antidote treatment includes atropine and pralidoxime [5]. The antimuscarinic medication of atropine is the basic antidote with proven efficacy for OP intoxication. The iv administration of atropine to the patient was given in the emergency service and intensive care. Without pharmacological intervention the organophosphate-cholinesterase bond does not spontaneously reverse and if this continues for 24-48 hours, irreversible destruction of cholinesterase occurs. This is given the name "aging". Aging is a situation where an enzyme is not activated spontaneously or linked to the oxime and bound to the phosphoryl group permanent chemical changes occur. Aging occurs at difference times depending on the agent. Relief from aging is only provided by new enzyme production. Depending on how rapid aging is, reactivation treatments are inversely effective. For oximes to be effective, they need to be administered before this process, and should being within 7-48 hours at the latest [10]. In our case pralidoxime was administered 3 hours after admission to the emergency service. The mortality rate linked to organophosphate and carbamate intoxication is linked to factors such as the material taken, amount, previous health situation, time since being found or transported, respiratory support, intubation and separation from the ventilator and incidence is mean 3-25% [7]. Our case was a severe intoxication case, who had mortal progression in spite of administered treatment.

Conclusion

Organophosphate compounds are commonly used in many areas, are easily accessed for suicide attempts, mistakenly used in pediatric and psychiatric disorder cases with exposure in farmers. A high rate of exposure to these compounds is more toxic. The mechanism of effect is though inhibition of the cholinesterase enzyme in the nervous system. In patients in the risk group taken to emergency service with consciousness disorders and cholinergic symptoms, intoxication should be considered. Specific treatment of atropine and pralidoxime should be administered in the shortest time. Decontamination should be performed depending on route of exposure without loss of time. In those with symptoms of systemic intoxication, mechanical ventilation support should be provided.

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