Case Report

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From Symptom to Diagnosis: A Case of Organophosphate Poisoning in an Unconscious Paediatric Patient

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Abstract

Organophosphates are used as pesticides in agriculture and poisoning is widespread all over the world. Organophosphates inhibit the enzyme cholinesterase which breaks down acetylcholine in the nervous system. As a result of this inhibition, acetylcholine accumulates in the central nervous system, autonomic ganglia and nerves. This leads to cholinergic discharge. The clinical picture depends on cholinergic discharge and varies from nonspecific symptoms to coma. Mortality and morbidity are high. A 3.5-year-old male patient was brought to the emergency department unconscious. On initial evaluation, he had a Glasgow coma scale of 10 and concomitant myosis and bradycardia. Haemogram, blood electrolytes, blood glucose, brain tomography and electrocardiogram were normal. In his medical history, it was learnt that he had contact with organophosphate inhalation. Procedure and treatment were planned. The diagnosis of organophosphate poisoning is based on medical history and clinical findings and there is no specific laboratory test. Therefore, it is vital to make a differential diagnosis in patients presenting with coma and confusion. In our patient, the diagnosis of organophosphate poisoning was understood after a detailed medical history and we would like to emphasise the importance of medical history in the differential diagnosis.

Keywords: Bradycardia, child, poisioning, unconscious

Introduction

Organophosphates are generally used as pesticides in agricultural fields (1). Although organophosphates have many advantages in increasing agricultural yield, they are potentially dangerous for humans and other living organisms in the environment (1). Poisoning is observed as a result of suicidal use and accidental exposure. In these cases, mortality and morbidity rates are high (1,2).

Although it is difficult to determine the actual incidence of organophosphate poisoning due to the difficulties in data collection, it is estimated that pesticide poisoning causes 250.000 - 350.000 deaths per year globally (3). Organophosphate pesticides are recognised as the largest group of pesticides used globally for agriculture, animal husbandry and other commercial purposes (1,2).

The mortality rate in organophosphate poisoning is variable. It has been reported to be 3-25% on average depending on the substance ingested, amount, previous health status of the patient, factors related with respiratory support, intubation and weaning from the ventilator (2,3).

Starting appropriate treatment by making a differential diagnosis is very important for patient mortality (3,4). As in all paediatric patients, medical history and examination findings are very valuable in the diagnosis in patients in whom intoxication is considered. Here, organophosphate intoxication detected by detailed medical history interrogation in a paediatric patient who was brought to the emergency department in an unconscious state will be presented.

Case Report

A 3.5-year-old male patient who was known to be healthy was brought to the emergency department unconscious. It was learnt that he suddenly started to fall asleep while his hair was being cut at the barber just before his admission and then did not respond to sounds. The patient's medical history revealed that he had no fever, had eaten at home with his family 1 hour ago, and had no history of trauma or infection in the last 1 month. It was learnt that neuromotor development was compatible with his age, there was no history of convulsion, no consanguineous marriage, no known diabetes, cardiac or neurological disease in the family.

In the initial evaluation of the patient; peak heart rate was 70/min, SPO was 98%, respiratory rate was 28/min and blood pressure was 85/65 mm/Hg. On neurological

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examination, Glasgow Coma Scalewas 10 and bilateral myosis was found in the pupils. Lateralisation findings and nuchal rigidity were not detected. Other system examinations were evaluated as normal.

Blood tests revealed fingerstick blood glucose 95 mg/dl, creatinine 0.26 mg/dl, sodium: 138 potassium: 3.9 AST: 28 ALT: 11 Hb: 12,4 leucocyte: 9000 platelet: 478.000 and was evaluated as normal. No acidosis, alkalosis or lactate elevation was found in blood gas. Brain tomography was found to be normal. Electrocardiogram was compatible with sinus bradycardia.

Intoxication was considered because of pupillary miosis and sinus bradycardia accompanying loss of consciousness. When he was evaluated with toxidrome findings, his medical history of organophosphate poisoning was questioned again. It was learnt that the patient sat next to the beans at his father's workplace just before going to the barber, tried to put them in his mouth once, but his father prevented him from doing so, and he put them in his mouth and took them out immediately. It was learnt that the beans were sprayed with tebuconazole-containing organophospate pesticide.

During this period, the patient, who had spontaneous breathing and was monitored, regressed his tendency to sleep and his Glasgow Coma Score was evaluated as 15.

The patient was consulted to 114 National Poison Advisory Centre. It was recommended to follow-up for 24 hours in terms of cholinergic symptoms. Cholinesterase level could not be evaluated in our hospital. In the ward follow-up, no acute pathology was detected and the patient was discharged with healing. Outpatient follow-up is continued due to the risk of possible chronic effects.

Discussion

Acute organophosphate poisonings which occur as a result of suicide or accident are frequently observed in our country, especially in rural areas, as in the whole world (3).

The mechanism of action of organophosphates is through inhibition of cholinesterase enzyme in the nervous system (3,4,5). Acetylcholine is the main neurotransmitter in the autonomic and somatic nervous system (4). Cholinesterase hydrolyses acetylcholine to choline and acetic acid. Inhibition of cholinesterase by organophosphate absorption leads to acetylcholine accumulation and excessive stimulation. This leads to cholinergic discharge in the central nervous system, autonomic ganglia, parasympathetic and sympathetic nerves. Symptoms caused by cholinergic discharge in the central nervous system include anxiety, insomnia, emotional lability, tremor, headache, dizziness, mental confusion, delirium and hallucinations. With nicotinic (sympathomimetic) action, muscle fasciculations, muscle weakness, hypertension and tachycardia may occur. The muscarinic effect causes smooth muscle contractions in all organs (e.g. lungs, gastrointestinal tract, eyes, bladder, secretory glands). This may result in salivation, lacrimation, sweating, myosis, urinary incontinence, and bradyarrhythmia, or it may cause weakening of sinus node and AV node conduction resulting in ventricular dysrhythmias. Symptoms and signs develop depending on the balance between nicotinic and muscarinic receptors (4,5). In our patient, myosis due to muscarinic effect, bradycardia and loss of consciousness due to central nervous system cholinergic discharge were observed.

The clinical evoliotion depends on the agents used, the amount of absorption and the mode of exposure (3,4,5). Onset of symptoms is very rapid with inhalation and slowest with transdermal absorption (3,4,5). In our patient, absorption occurred both by inhalation and gastrointestinal route and symptoms appeared in approximately 1 hour.

Most patients start to show symptoms within 8-24 hours after ingestion, depending on the amount ingested (3,4,5). Patients should be monitored in terms of coma, seizure, respiratory failure, excessive secretions or severe bronchospasm (5,6). The need for endotracheal intubation may arise due to bronchospasm (5,6).

The treatment process after poisoning consists of decontamination, prevention of absorption, general support and intensive respiratory support (5,6). Atropine forms the basis of treatment in organophosphate poisoning because of its anticholinergic properties. Atropine was not administered in our patient because bradycardia did not persist. Antidotes are administered according to the degree of intoxication. As an antidote, pralidoxime (PAM) is a cholinesterase reactivator which helps to reverse bronchospasm and muscle fasciculation by accelerating the restoration of enzyme activity in the neuromuscular junction (5,6). No antidote was administered in our patient because of the absence of bronchospasm findings.

Exposure to organophosphate may cause free radical production and consequently lipid peroxidation, which may lead to DNA damage (7,8). For children, organophosphate exposure has been associated with developmental problems such as decreased IQ level and attention deficit hyperactivity disorder (7,8). In a study by Chbara et al. the risk of organophosphate-induced delayed neurotoxicity was reported (9). Delayed neurotoxicity is a rare condition that may occur weeks after the first exposure and is a distal axonopathy affecting both central and peripheral nervous system. It manifests as motor-sensory polyneuropathy in the peripheral nervous system and myelopathic symptoms in the central nervous system (9). In organophosphate poisoning, management of chronic findings as well as acute findings is important in terms of morbidity (9). Our patient was followed up in terms of possible chronic symptoms.

The differential diagnoses of intoxication in unconscious paediatric patients include endocrine and metabolic causes, trauma, seizure, vascular pathologies, cardiac diseases, infections and psychiatric convulsions (10).

Organophosphate intoxication must be kept in mind in

the differential diagnosis in patients who present with altered consciousness and in whom anamnesis cannot be obtained, if there are findings related with nicotinic and muscarinic effects.

Conclusion

Organophosphate poisoning is a public health problem with high mortality and morbidity. It is vital to make a differential diagnosis and to provide appropriate intervention in the early period. Clinically specific findings may not always be observed in patients. Especially in patients who are brought to the emergency department unconscious as in this case, it may be more difficult to make a differential diagnosis. For this reason, diagnostic medical history and examination findings are very valuable in patients with intoxication as in all paediatric patients. In our patient, organophosphate poisoning was determined by taking a detailed medical history.

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