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Case report

Organophosphate poisoning due to transdermal application of diazinon in a 7-years-old child

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Abstract

Organophosphate compounds remain a major cause of pesticide-related intoxications worldwide. Their toxicity results from inhibition of acetylcholinesterase, leading to accumulation of acetylcholine at synaptic junctions and overstimulation of nicotinic and muscarinic receptors.

We report a rare case of severe organophosphate poisoning following transdermal exposure in a seven-year-old girl. The patient developed altered consciousness and generalized seizures after topical application of an agricultural insecticide for the treatment of scalp pediculosis. She received prompt symptomatic and antidotal therapy, with a favorable clinical outcome.

This case highlights the potential severity of dermal exposure to organophosphates in children and underscores the importance of public awareness regarding inappropriate domestic use of agricultural pesticides.

Keywords: Organophosphate, poisoning, transdermal application, Diazinon, pesticide.

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1. Introduction

Organophosphate compounds constitute a heterogeneous group of chemicals whose basic structure was first described by Schrader. Structurally, these agents are characterized by a central phosphorus atom bonded to two organic radicals (R1 and R2), a double bond to either oxygen or sulfur, and a variable functional group (X), which allows them to be classified into four groups (Nisse, 2012).

Group 1 includes compounds in which X is a quaternary ammonium group;

Group 2 comprises compounds with a fluorine atom as the substituent;

Group 3 includes compounds in which X is OCN, CN, or SN. These three groups mainly correspond to nerve agent weapons.

Group 4 includes all other organophosphates with different substituents.

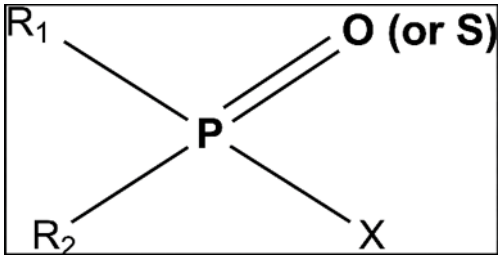

Figure 1: general structure of organophosphate agent (Costa, 2006)

We report a case of an unusual case of organophosphate intoxication resulting from transdermal absorption in a seven-year-old child, following the inappropriate application of an agricultural insecticide (diazinon) to the scalp for the treatment of pediculosis. The product was applied for approximately nine hours, resulting in severe systemic toxicity.

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2. Case report

A seven-year-old girl with no previous medical or surgical history was admitted to the emergency department of Hassan II University Hospital in Fez for generalized tonic–clonic seizures occurring approximately two hours after topical application of Diazinon 60% (Figure 2) to the scalp. The amount of powder used was unknown. The mother had diluted the product in water to obtain a thick mixture, which was applied to the child's head overnight.

Several hours after application, the patient developed impaired consciousness followed by epileptic seizures. No other signs were initially observed, particularly those suggestive of a muscarinic syndrome. A detailed history obtained from the father revealed that the application occurred at around 3 a.m. The child had been suffering from scalp pediculosis, and the mother attempted eradication using an agricultural organophosphate insecticide.

On admission, the patient was unconscious with a Glasgow Coma Scale score of 9. Cranial nerve examination and osteotendinous reflexes were normal. Pupils were markedly constricted. The patient was placed under continuous monitoring of hemodynamic, cardiopulmonary, and neurological parameters and underwent extensive scalp decontamination.

Due to neurological and respiratory deterioration (SpO_2 65%), endotracheal intubation and mechanical ventilation (FiO_2 50%) were initiated, along with sedation. Antidotal therapy with atropine and pralidoxime methylsulfate was administered. Toxicological analysis revealed markedly decreased cholinesterase activity (0.2 KU/L). Qualitative analysis by thin-layer chromatography confirmed exposure to an organophosphate compound.

Clinical evolution was favorable. After 10 days, cholinesterase activity progressively increased (2.6 KU/L on day 7 and 3.1 KU/L on day 10), and the patient achieved complete clinical recovery.

Figure 2: Picture of Diazin-60% flacon used in the transdermal application.

3. Discussion

Organophosphate pesticides were developed as alternatives to organochlorine compounds. Organochlorine compounds are composed of a carbon backbone bonded to several chlorine atoms; consequently, their molecular weights are considerable. They were widely used in the late 19th and early 20th centuries but later abandoned due to their toxicity and environmental persistence (Baert and Danel, 2004).

Organophosphate agents are responsible for the genesis of an acetylcholine hyperstimulation toxidroma because of the phosphorylation of the active site of the enzymes involved in the catabolism of organophosphate agents (acetylcholinesterase normal values: 4-10 female, 5-11 men), consequently the amount of acetylcholine in the synaptic area increases considerably (Hachelaf et al., 2006). Besides the main toxic action of diazinon to inhibit acetylcholinesterase activity, it is established that some organophosphates (including Diazinon) inhibit different ATPases, the group of enzymes playing an important role in biochemical processes, as a result, cellular damages of depletion of the energetic metabolism occurs, with liberation of free radicals and other toxic metabolites (Čolović et al., 2010; Krstić et al., 2010).

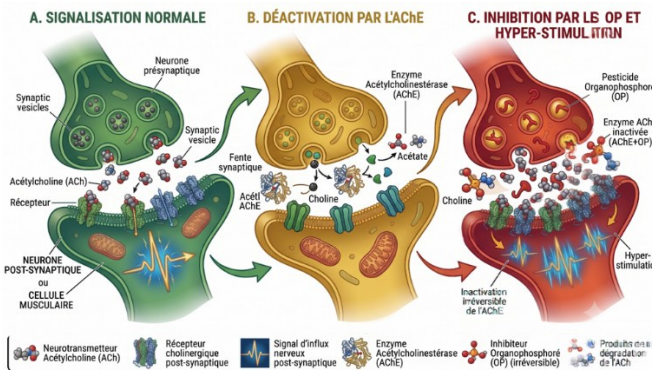

Figure 3: Mechanism of action of organophosphate compounds.

The diagnosis of intoxication by an organophosphate agent is clinical and biological; clinically, hyperstimulation of cholinergic receptors leads to hypersecretion of glands, fasciculation, and cramps. The extreme gravity results from the diaphragm alteration, which could be responsible for respiratory failure.

The main analytical tools in disposition of laboratories in terms of confirming an exposure to organophosphate agents are represented by calculating the cholinesterase activity in blood samples, which represents an indirect test to determine such a condition, by their combination to cholinesterase calculating the remaining portion of cholinesterase is an important indicator of intoxication (Jalady and Dorandeu, 2013).

Therapeutic measures in organophosphate intoxication consist of symptomatic treatment, which consists of correction of the main vital failures, such as

cardiopulmonary function, neurologic status, gastric decontamination, and activated charcoal, and eventual administration of antidotic therapies consisting of pralidoxime and atropine.

Diazinon is an organophosphate compound belonging to the thiophosphoryl class. Its commercialization started in 1952 by Ciba-Geigy, a Swiss chemical manufacturer (Burgess et al., 2008). It is known to be an indirect organophosphorous agent as well as the famous Parathion, which is activated by oxidation to diazinoxon, a very potent cholinesterase inhibitor, with both the parent compound and diazoxon inactivated by hydrolysis to their corresponding phosphoric acid derivatives: diethylphosphorothioic acid and diethylphosphoric acid, respectively. Minor metabolites include hydroxydiazinon, formed by oxidation of the isopropyl side chain. This metabolite is toxic and is found in the blood at levels equivalent to 25-70% of the parent compound. Diazinon accumulates in fat in concentrations over 100 times those found in blood. In experimental animals, more than 80% of a dose is eliminated as the hydrolysis products in the urine (24 h) (Feldmann and Maibach, 1974).

The human cases of exposition to pesticides agent by transdermal path remain as sporadic cases (including organophosphate agents), by the second half of the 20th century researchs were focused on the establishment of a pattern of causality between an history of exposition to organophosphate (or other similar compounds) and the emergence of a lot of diseases (experiments were made on animal models), the reasons behind these studies were the notorious cases of diseases in population exposed in professions (agriculture and soldiers), the establishment of the causality were affirmed statistically and objectively, furthermore the studies were directed then to the determination of the limits of the apparition of these health injuries due to the chronic exposition to these compounds in order to the amelioration of conditions of workers exposed to these compounds.

The cutaneous barrier is histologically constituted by epidermis, dermis, and hypodermis; the epidermis is made of germinative, spinanosum, granulosum and corneum stratums.

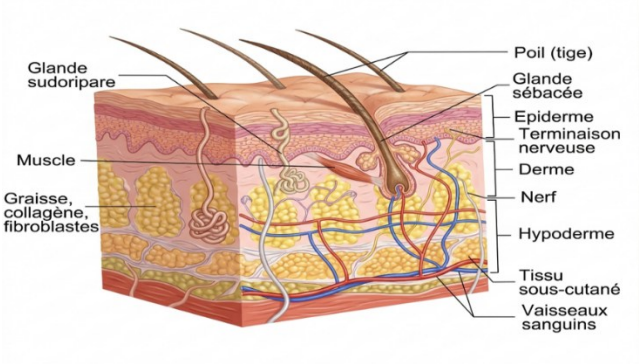

Figure 3: cutaneous stratification and its cellular composition.

The major works led to studies of the cutaneous passage of xenobiotics in animal models; human skin is a more effective barrier to penetration than that of certain small animals. The face, forehead, scalp, and neck absorb2-6 times more than the forearm. These areas are most likely to be exposed. The hands and feet absorb about as much as the forearm does, but the absorption is slower. Larger areas of the body, such as the back, allow greater absorption than the forearm. Other parameter sinfluences the percutaneous passage of pesticides, such as the chemical properties of the agent, a high lipophily and hydrosolubility of the compound allow an increased percutaneous passage, therefore the adjunction of solvent into the commercial preparation leads to enhancement of the cutaneous passage and in infants (The thin skin barrier) (Boudry et al., 2008). The main analytical tools for the disposition of laboratories in confirming exposure to organophosphate agents are the measurement of cholinesterase activity in blood samples, which is an indirect test for this condition. By combining the cholinesterase, calculating the remaining portion of cholinesterase is an important indicator of intoxication (Čolović et al., 2010).

Degradation of acetylcholineisleaded by two enzymes acetylcholinesterasefoundedescentially in central nervous system, the circulating of this enzyme butyrylcholinesterase, the catabolism of acetylcholineliberatetwo catabolites aceticacid and choline, the choline isrecapted in pre-synaptic area by transporterscalledVachT (acetylcholinetransporters) to regenerateacetylcholine by choline acetyltransferase (CAT) (Kobayashi et al., 2011), furthermore acetylcholine is degraded by acetylcholinesterase researchs doesn't mentionned any certain physiological function of the other enzyme, thus an increasment of the concentration of medicines such as succinylcholine have been documented in genetic phenotype whith an unfunctionnal butyrylcholinesterase (Lockridge et al., 2011). The therapeutic attitude in front of an organophosphate intoxication is based on symptomatic treatment which aim to establish the vital functions of human body (cardiocirculatory, respiratory and neurological, hepatic, renal and metabolic functions); gastric lavage and the antidotic treatment including administration of atropine and methylsulfate of pralidoxime, the muscarinic and nicotinic receptors are highly stimulated for this purpose administration of atropine is important therefore the muscarinic symptoms including the generalized glandular secretion are stopped; furthermore the methyl sulfate of pralidoxima regenerate acetylcholinesterase and it has an important implication in stopping the nictonic syndrome (Kouraichi and Thabet, 2008).

Neurological disorders are present in the majority cases of organophosphate intoxication, they englobe several degree of perturbations passing from minor alteration of the consciousness status to a profound coma, epileptic seizures, recently some advancements in pharmacology leads to the synthesis of analogues of neurosteroids, they are many neurosteroids found in the brain such as allopregnanolone (brexanolone), pregnanolone, androstanediol, and allotetrahydrodeoxycorticosterone, Neurosteroids act as

positive allosteric modulators and direct activators of GABA-A receptors, they decrease the neuroexcitability considerably rather than benzodiazepines or barbiturics (Reddy, 2019).

As described, atropine and pralidoxime methylsulfate have long been the mainstay antidotes for organophosphate intoxication; recent discoveries have generated hope for reduced mortality from these agents.

The new approach to the treatment of organophosphorous intoxication consists of a panel of molecular antidotes called enzymatic antidotes, which trap, inactivate, or catabolize many toxins, including organophosphorous compounds. The mechanistic of the bioacavengersconsist on a first time into the trapping of organophosphates compounds (these molecules act like the mechanistic of recognizing of antibodies), after their incorporation another enzymes degrade them, nonetheless these enzymes are not in mesure of the reactivation of enzymes responsible of the degradation of acetylcholine; nowadays it exists some combination of those new compounds to the methylsulfate of pralidoxime in order to enhance the efficacity by a simultaneous action of degradation the toxic and regenating enzymes of degradation (Jacquet et al., 2019).

4. Conclusion

Organophosphates are toxic compounds which should be manipulated carefully. Education for agricultural workers is important. They must understand the risks, know how to read safety labels, and comply with re-entry intervals for treated areas. Employers are legally required to provide this training. The indoor use of these compounds ought be avoided especially the presentations reserved for professional purposes.

Data availability statement

The original contributions presented in the study are included in the article/Supplementary Material; further inquiries can be directed to the corresponding authors.

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Conflict of interest

The authors declare that the research was conducted without any commercial or financial relationships that could be construed as a potential conflict of interest.

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