

Subcutaneous Injection of Organophosphate Parathion: An Unusual Way of Intentional Acute Poisoning

Hanane Ezzouine, Mahmoud Kerrous, Saad El Haoui, Soufiane Ahdil and Abdellatif Benslama

Medical Intensive Care Unit, University Teaching Hospital, Ibn Rushd, Casablanca, Morocco

Corresponding author: Hanane Ezzouine, Faculty of Medicine and Pharmacy, University Hassan II, Casablanca, Morocco, Tel: 00212619918090; Email: ezzouinehanane@yahoo.fr

Received date: July 21, 2016; **Accepted date:** September 28, 2016; **Published date:** October 05, 2016

Citation: Ezzouine H, Kerrous M, El Haoui S, et al. Subcutaneous Injection of Organophosphate Parathion: An Unusual Way of Intentional Acute Poisoning. J Med Toxicol Clin Forens Med. 2016, 2:2.

Copyright: © 2016 Ezzouine H, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Abstract

The oral way method is the mostly used and well-known way recognized in the intentional acute organophosphate poisoning. The peculiarity of the case we report is the subcutaneous injection route and its evolution.

A 25 year old patient was admitted to intensive care for respiratory distress which was complicated by an intentional subcutaneous injection of parathion "METYPHON50[®]" two days before. The immediate evolution was marked by asthenia, progressive deterioration of general condition and vomiting. After his hospitalization on his third day of poisoning, the patient was comatose, had a tight myosis and a diffuse tremor hypersalivation. His hemodynamic parameters were correct. He had respiratory distress and a right upper limb swollen and inflamed mainly at the injection site (inner left arm). The anticholinesterase activity was 12.5% and his cerebral CT was normal. The therapeutic management was intubation and ventilatory support added to symptomatic treatment. The contrathion could not be administered because it was not available. The evolution was favorable on the third day of hospitalization with regression of toxidrom and patient extubation. The activity of acetylcholinesterase was 50% at control. The acute intoxication by the subcutaneous route to organophosphates was exceptional. Pharmacodynamics and kinetics of the product were involved in the clinical expression throughout the duration of elimination. It is important to identify the product characteristics organophosphate offending. The symptomatic treatment is always to put more and to use no antidote as in our case.

Keywords:

Subcutaneous injection; Organophosphate poisoning; Parathion; Prognosis

Introduction

Organophosphates poisoning is an important occupational health issue because they are pesticides worldwide used in

industries and agriculture and in different job functions can lead to different levels of exposure. Most occupational exposures are caused by absorption through exposed skin. This exposure is sometimes enhanced by inhalation in settings including spraying operations. Most cases of intentional pesticide poisoning appear to be impulsive acts undertaken during stressful events, and the availability of pesticides strongly influences the incidence of self-poisoning. Acute intoxications represent 35% of admissions in the medical intensive care unit of University Teaching Hospital Ibn Rushd at Casablanca in Morocco. Organophosphate poisonings come second with 20%. The intentional oral route is the most frequently followed? The peculiarity of this observation is the subcutaneous injection route and its evolution.

Organophosphate compounds (OPs) have been used as pesticides many of the methods used currently for the detoxification of OPs are harmful and possess serious environmental consequences. Therefore, utilizing enzymes for the detection and decontamination of OPs is gaining increasing attention as an efficient and clean bioremediation. Microbial enzymes, such as OP hydrolases, OP acid anhydrolases or methyl parathion hydrolase (MPH), are potent agents for OP decontamination [1].

Case Report

We report the case of a 25 year old patient who was admitted to intensive care in the aftermath of a self-subcutaneous injection of methyl-parathion "METYPHON[®] 50" with suicidal intent. The family reported that he have had injected himself the compound. In the immediate aftermath, he had general malaise, vomiting and asthenia with a progressive deterioration of general condition evolving for three days. On the fourth day, he presents consciousness disorders.

Upon ICU admission, Glasgow scale was 7/15. He had bilateral pin point pupils without signs of motor weakness as paraparesis or paraplegia. However, he had diffuse tremors and excessive salivation. His blood pressure was 120/50 mm Hg and heart rate to 127 beats/min.

The electrocardiogram showed repolarization disorders in type of anterior and septal negative T-waves. His respiratory rate was 14 cycle/min. His arterial oxygen saturation was 87% in the open air. Pleuropulmonary auscultation found snoring rales in lung bases. Clinical examination found, moreover, a swollen left upper limb to the axilla, red and hot to the touch necrosis and infiltration at the injection site on the left elbow with an evocative appearance of cellulitis.

The biological assessment scored normal renal function, blood urea at 0.30 g/l and creatinine to 11 mg/l. serum sodium was 140 mEq/L and serum potassium to 4.2 mEq/l. Transaminases were normal; TGO to 6 IU/L and TGP 10 IU/l. Troponins were below 0.02 ng/ml. The plasma cholinesterase activity was 12.5% on the first day of hospitalization, the fifth of intoxication. Chest radiography found a right basal parenchymal lung disease. The CT scan was normal.

Our therapeutic management was intubation and mechanical ventilation, antibiotics to treat aspiration pneumonia, symptomatic treatment of muscarinic signs using Atropin 1 mg per 15 min until they disappear, a clinical and laboratory monitoring. The evolution was marked by a neurological improvement with the disappearance of miosis on the fifth day of hospitalization. The pralidoxime could not be administered as it was not available. The evolution was favorable to the third day of hospitalization with regression of toxidrom and patient extubation on the sixth day of hospitalization. Control of cholinesterase activity was 50% and the patient was discharged from the intensive care unit after psychiatric counselling.

Discussion

The injectable route is an uncommon way to organophosphate poisoning [2, 3]. The route of poisoning, the toxic nature of the product and its mechanism of action are the major prognostic parameters involved in the symptomatic clinical and laboratory expression [2-4]. The injectable mode is exceptional in organophosphate poisoning [2-4].

The pharmacodynamic properties of some of these products, including methyl parathion and the kinetics of cholinesterase activity in the brain, parallel to the plasma activity depend on the route of administration of the toxicant [5].

Parathion is activated by its metabolite paraoxon. Plasma cholinesterase activity of methyl parathion is time-dependent on the route of the toxic administration [6, 7]. *In vivo*, the intravenous or oral administration of 2.5 mg/kg of methyl-parathion, drop it in under 60 min and the reactivation of cholinesterase is 30-48 h *in vitro*. The spontaneous reactivation of cholinesterase is complete after 6 h at 37°C [5]. Dermal inhibition of cholinesterase activity of methyl parathion is dose dependent. It develops slowly and lasts 6 h of 48 pm. *In vivo*, when administered intravenously or orally methyl-parathion cholinesterase reactivation may partially reflect the spontaneous reactivation suggesting a rapid clearance of methyl parathion or its active metabolite, methyl paraoxon. More gradual and prolonged cholinesterase inhibition, after dermal exposure is related to the location of toxic methyl-parathion or its metabolite in a site in which their distribution is slower and

exhibited by these facts at major risk of prolonged side effects [4, 6, 7]. Expression of cholinesterase activity of methyl parathion in the brain and other organs in parallel with its activity in plasma depends on the route of administration of the product, its half-life and its distribution [5]. All these data suggest that the chronological clinical expression in the case reported is due to the prolonged toxic effects of methyl-parathion associated to dermal and subcutaneous absorption. The main signs were unconsciousness as in brain manifestation, miosis, vomiting, diffuse tremors and excessive salivation. All these signs delayed more than 48 h after subcutaneous injection of methyl-parathion which half-life is within this range.

Conclusion

The pharmacodynamic properties of methyl parathion depend on the mode of exposure to the product [5-8]. Reactivation of plasma cholinesterase is slower in case of poisoning by dermal even subcutaneous way, modulated by the location and site distribution of methyl parathion and its metabolite [6, 7]. The acute intoxication by the subcutaneous route to organophosphates is exceptional. Pharmacodynamics and kinetics of the product are involved in the clinical expression, throughout the duration of elimination. It is important to identify product characteristics organophosphate offending. The symptomatic treatment is always to put more and to use no antidote as in our case.

References

1. Schenk G, Mateen I, Ng TK, Pedroso MM, Mitić N, et al. (2016) Organophosphate degrading metallohydrolases: Structure and function of potent catalyst for applications in bioremediation. *Coord Chem Rev*.
2. Volk O, Reith S, Saehn C, Haars P, Bach D, et al. (2002) Attempted suicide by intravenous injection of Metasystox®. *Anästhesiol Intensivmed Notfallmed Schmerzther* 37: 280-283.
3. Guven M, Unluhizarci K, Goktas Z, Kurtoglu S (1997) Intravenous organophosphate injection: An unusual way of intoxication. *Hum Exp Toxicol* 16: 279-80.
4. Qiao GL, Williams PL, Riviere JE (1994) Percutaneous absorption, biotransformation and systemic disposition of parathion *in vivo* in swine I. Comprehensive pharmacokinetic model. *Drug Metab Dispos* 22: 459-471.
5. Kramer RE, Wellman SE, Zhu H, Rockhold RW, Baker RC (2002) A comparison of cholinesterase activity after intravenous, oral or dermal administration of methyl parathion. *J Biomed Sci* 9: 140-148.
6. Wester RC, Quan D, Maibach HI (1996) *In vitro* percutaneous absorption of model compounds glyphosate and malathion from cotton fabric into and through human skin. *Food Chem Toxicol* 34: 731-735.
7. Zhu H, Rockhold RW, Baker RC, Kramer RE, Ho IK (2001) Effects of single or repeated dermal exposure to methyl parathion on behavior and blood cholinesterase activity in rats. *J Biomed Sci* 8: 467-474.
8. Abel EL, Bammler TK, Eaton DL (2004) Biotransformation of methyl parathion by glutathione S-transferases. *Toxicol Sci* 79: 224-232.