

Case Report

Acute poisoning in a child following topical treatment of head lice (pediculosis capitis) with an organophosphate pesticide

Muddathir H Hamad (1), Ahmed Awad Adeel (2), Ali Abdu N Alhaboob (3), Ahmed M Ashri (3),
Mustafa A Salih (1)

(1) Division of Pediatric Neurology, (3) Pediatric Intensive Care Unit, Department of Pediatrics, (2) Department of Microbiology and Pathology, College of Medicine and King Saud University Medical City, King Saud University, Riyadh, Saudi Arabia

ABSTRACT

This is a case report of acute organophosphate poisoning in a child treated with topical application of Diazinon-60 (WHO Class II toxicity) for head lice (pediculosis capitis). The patient presented with neurological symptoms and signs. After emergency respiratory and circulatory resuscitation the patient underwent dermal decontamination and was treated with atropine, high flow oxygen and pralidoxime. Scanning electron micrographs of scalp hair specimens revealed both viable and empty head lice nits (lice eggs that attach to the hair shaft). The patient

was hospitalized for seven days and discharged after full recovery. The case highlights the importance of raising the awareness of health workers and the community about the danger of misusing pesticides for the treatment of head lice.

Keywords:

Organophosphate poisoning; Children; Head lice; Pediculosis; Diazinon; Head lice nits; Scanning electron microscopy

Correspondence to:

Dr Muddathir H Hamad,
Division of Pediatric Neurology, Department of Pediatrics,
King Khalid University Hospital and College of Medicine,
King Saud University, P O Box 2925,
Riyadh 11461, Saudi Arabia.
Email: mudhamad@yahoo.com

How to cite this article:

Hamad MH, Adeel AA, Alhaboob AAN, Ashri AM, Salih MA. Acute poisoning in a child following topical treatment of head lice (pediculosis capitis) with an organophosphate pesticide. Sudan J Paediatr 2016;16(1):63 - 66.

INTRODUCTION

The World Health Organization (WHO) estimates the incidence of pesticide poisoning to be three million cases per year, leading to more than 250,000 deaths annually [1,2]. Organophosphate insecticides are a group of insecticides acting on the enzyme acetylcholinesterase. They irreversibly inactivate this enzyme which is essential to nerve function in insects, humans, and many other animals and affect this enzyme in variable ways and thus in their potential for poisoning [3]. Pesticide regulators and health workers are concerned that a number of organophosphate insecticides are commonly used agriculturally on fruits and vegetables and for control of pests such as mosquitos in public spaces. They can pose a serious health hazard if they are not used according to pesticide regulations that guard against their health hazards. The present paper reports a case of misuse of an organophosphate pesticide leading to acute organophosphate poisoning.

CASE REPORT

A 7-year-old girl, known to have bronchial asthma on budesonide inhalation and salbutamol, was brought to King Khalid University Hospital, Riyadh with vomiting, dizziness and altered level of consciousness. Past medical history revealed episodes of syncopal attacks that started by the age of 5 years, occurring 1-2 times per year and lasting for few seconds. Her symptoms started 15 minutes after she applied Diazinon-60® (an organophosphorus compound, used for control of ectoparasites in animals) to her hair for treatment of lice. Upon arrival to the emergency room, she was found to be lethargic with Glasgow Coma Scale (GCS) of 14, pinpoint pupils and dry mouth. Her pulse rate was 105/minute; respiratory rate was 19 breaths/minute and blood pressure of 95/69 mmHg. She had normal muscle tone, power, and reflexes in the upper and lower extremities. Other systems examination was normal. The airway was

secured, and high flow oxygen was given, and the patient was attached to cardiac monitor. She received one dose of atropine (0.05 mg/kg) and one dose of pralidoxime (20 mg/kg) and shifted to the Pediatric Intensive Care Unit (PICU) for 24 hours. She received pralidoxime at a rate of 10 mg/kg/hour as a continuous infusion for 16 hours and intravenous atropine hourly for 4 hours, then 2 hourly for 2 hours. All laboratory investigations were normal including complete blood count (CBC), venous blood gas (VBG), renal and liver function tests. ECG and Holter monitoring showed no evidence of arrhythmias. Cholinesterase activity was not measured. Electroencephalogram (EEG) and brain magnetic resonant imaging (MRI) were unremarkable. Scanning electron micrographs of scalp hair specimens revealed both viable and empty head lice nits (lice eggs that attach to the hair shaft) [Figures 1 and 2]. She was discharged after 7 days. Her subsequent follow up visits showed normal neurological examination.

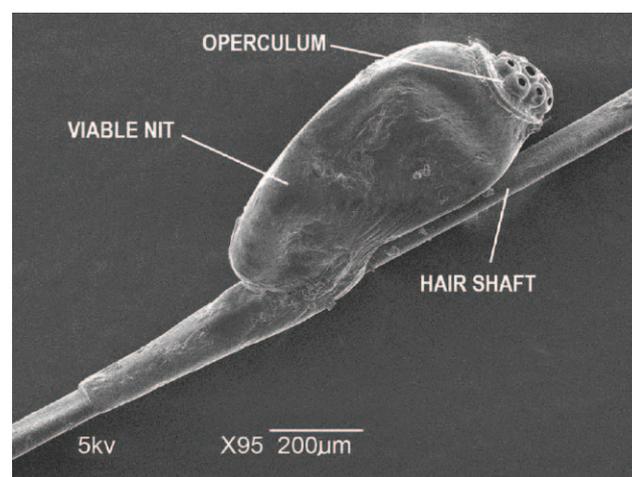


Figure 1 - Scanning electron micrograph of a viable nit on the patient's hair shaft. Under light microscope a moving embryo may be seen inside the nit and a prominent eye could also be noted.

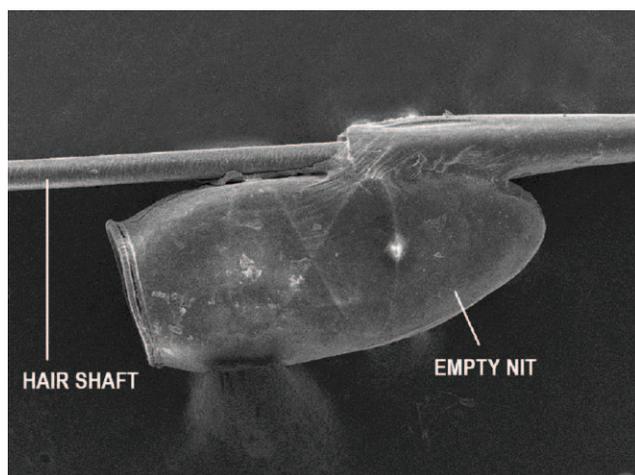


Figure 2 - Empty nit on the patient's hair shaft revealed by scanning electron microscopy. Note the missing operculum after release of embryo. The empty nit looks transparent under light microscope.

DISCUSSION

The recommended regimens for treatment of pediculosis capitis are : permethrin 1%, pyrethrin 0.33% and piperonyl butoxide 4%, malathion 0.5% lotion, benzyl alcohol 5% lotion , ivermectin 0.5% lotion and spinosad 0.9% topical suspension [4]. Of these, only malathion is an organophosphate. However, malathion is classified by WHO as Class III (slightly hazardous pesticide) whereas diazinon, used for the present case is classified as Class II (moderately hazardous pesticide)[5]. Even the slightly hazardous insecticides are usually used with caution when applied for pediculosis [6]. Diazinon is absorbed through the skin but occupational exposure of workers handling is prevented through the use of good work practices, gloves, coveralls, goggles, and other appropriate equipment [7]. It is clear that the application of this pesticide in this patient represents a case of pesticide misuse because it did not conform

to regulations.

The diagnosis of acute organophosphate poisoning depends on the history of exposure, clinical presentation, and laboratory investigations. The clinical presentation is variable and usually depends on the amount used, and the route of exposure .The clinical picture shows features of overstimulation of muscarinic acetylcholine receptors in the parasympathetic system, overstimulation of nicotinic acetylcholine receptors in the sympathetic system, overstimulation of nicotinic and muscarinic acetylcholine receptors in the central nervous system (CNS) and overstimulation of the nicotinic acetylcholine receptors at the neuromuscular junctions [8]. The common mode of presentation includes CNS symptoms which may include anxiety, confusion, seizures and coma. The muscarinic effects include diaphoresis, diarrhea, urination, miosis, bradycardia, bronchorrhea, bronchospasm, emesis, lacrimation and salivation. Other muscarinic effects may include muscle fatigue, fasciculation, paralysis, respiratory muscle weakness, tachycardia and hypertension. Cardiac arrhythmias, including heart block and QTc prolongation, are occasionally observed in organophosphorus agent poisoning [9]. Late onset polyneuropathy can occur following organophosphate poisoning [10,11]. The laboratory investigations may reveal low cholinesterase activity which confirms the diagnosis.

The first step in the management of organophosphorus poisoning is to assess the patient's airway, breathing, and circulation (ABCs). Endotracheal intubation may be needed to protect the airway. The patient should be attached to cardiac monitor. Circulatory support with intravenous fluids is crucial. Gastric decontamination is required in case of organophosphate ingestion. Dermal decontamination should be performed in the event of skin exposure to limit further absorption and to prevent contamination of health caregivers. Anticholinergic agents like atropine should be used in

frequent doses till the cholinergic signs are reversed [12]. Pralidoxime (2-PAM) is a cholinesterase reactivator used as antidote for organophosphate poisoning [13]. It restores respiratory and skeletal muscle strength, but it doesn't cross the blood brain barrier [14].

Pediculosis capitis is a common infestation in children in some parts of Saudi Arabia. One study reported the prevalence of 13.3% among school-age children [15]. The present case should call for raising the community awareness of the dangers of misusing pesticides for self-medication of pediculosis

particularly in children. Health workers should also be aware of the high risk of pesticide poisoning and should be trained on early diagnosis and appropriate management of cases of organophosphate poisoning.

ACKNOWLEDGEMENTS

The Authors wish to thank Dr. Omar Hamed Abd-Elkader, Associated Professor, Electron Microscope Unit, Zoology Department, College of Science, King Saud University for the elegant electron microscopy images.

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