Student's Journal of Health Research Africa
Vol. 4 No. 9 (2023): September 2023 Issue
https://doi.org/10.51168/sjhrafrica.v4i9.415
Original article

"WAS IT HIS MILK"? A CASE REPORT OF CHLORPYRIFOS AND CYPERMETHRIN MIXED POISONING IN A TODDLER.

Isaac Ebiju[a,b], Innocent Ayesiga[c,b], Godfrey Wekha[a,b,c,*], Julius Bruno Mugambe[d], Elias Musiime[b], Primrose Magala[e], Blaise Kiyimba[a]

[a] *Makerere University College of Health Sciences, Kampala, Uganda.*
[b] *Ubora Foundation Africa, Kampala, Uganda.*
[c] *Mbarara University of Science and Technology, Faculty of medicine Mbarara, Uganda.*
[d] *Kampala International Hospital.*
[e] *Moorfields Eye Hospital Uk.*

Abstract

Introduction

The prevalence of organophosphate poisoning has been increasing over the years, with significant hospitalization following the introduction of various pesticides. The inception of different regulating agencies against organophosphate utilization has been effective in developed countries, unlike low- and middle-income countries. Continuous monitoring of vulnerable populations, such as children, and depressed and anxious individuals, is necessary for reducing poisoning cases.

Case

We discussed a 2-year and 6-months old male child who was previously healthy and was brought into our pediatric emergency unit of a general hospital with a history of ingestion of fluid that he thought was milk. We present the sequelae of events from the ingestion of the substance until discharge from the facility. Additionally, we elaborate on the different preparations for managing any toddler with organophosphate poisoning.

Conclusion

Appropriate use of these compounds, instruction of the public about their harmful effects, and restriction of their uncontrolled sales by legal regulations can reduce the incidence of organophosphate poisoning. Promoting emergency management strategies among parents concerning the management of acute poisoning is vital in promoting better children's outcomes.

Recommendation

Heath regulation bodies must take an interest in educating health workers and the community concerning organophosphate poisoning and its management, especially in a limited resource setting.

Keywords: Chlorpyrifos, Cypermethrin, organophosphates, poisoning, mixed pesticide, Submitted: 2023-05-07 Accepted: 2023-06-06

1. Introduction.

According to WHO, organophosphate poisoning remains a significant cause of annual in-hospital admission at over 3 million cases globally, with an 8.3% case fatality proportion.1 Fol-

* Corresponding author.
Email address: wekha76@gmail.com (Godfrey Wekha)

lowing their discovery in the 1930s, they have increasingly gained popularity globally for use as pesticides.2 Research in developed countries has demonstrated an 18.2 per 100 000 full-time workers annual incidence of organophosphate poisoning among agricultural workers, with 7.4 per million cases among school children.2 The cumulative prevalence of poisoning has been contributed by both deliberate and accidental poisoning.3 Over 1 million cases have been estimated to be from accidental poisoning, while 2 million are deliberate, with occupational exposure as the most significant risk factor.4

Different regulating agencies have been installed to limit the utilization of organophosphates globally, for example, the Federal Insecticide Fungicide and Rodenticide Act (FIFRA), the United States Environmental Protection Agency (EPA), and Federal Food Drug and Cosmetic Act (FFDCA).5 However, the operation ability of these organizations has been limited to defined geographical locations hindering their regulation impact in other areas, for example, low- and middle-income countries, of which Uganda is inclusive.

In Africa, the volume of pesticides used is still lower, and the prevalence of organophosphate poisoning is 40-60% lower compared to other continents.6 Despite the prevalence in Africa, organophosphate poisoning is still a significant public health problem due to the lack of poison management centers and organophosphate market restrictions.

In Uganda, the prevalence of poisoning was 28.8%, with over 90% of admissions being due to organophosphate poisoning.7 Additionally, children were the most affected population following accidental ingestion, thus signifying higher vulnerability than other population categories.8 Systemic exposure to organophosphate toxins has been linked to the victim's pre-existing states, for example, being a toddler, depression, rejection, and severe anxiety. Individuals in these categories have a higher likelihood of organophosphate ingestion.9 Therefore, caution and attention must be directed to these individuals to reduce their exposure risk. The aim of this case report is to describe the chronology of events following an accidentally mixed pesticide (Cypermethrin and Chlorpyrifos) ingestion by a 2 -year and 6-month-old patient and a narrative of management events with their corresponding outcomes. We also provide information concerning the effective management of organophosphate poisoning in a toddler.

2. Case Report.

A 2 -year and 6-months previously healthy boy was brought in via the outpatient department to the pediatric emergency of a general hospital in central Uganda following accidental ingestion of a pesticide at around 10:00 am of 8th August 2022 while being used to spray cattle by a neighbor, thinking it was milk. Before admission, he asked the neighbor to offer him some water to drink while pointing at the bottle which contained the pesticide. This prompted Mr. E.P. to inquire, and he found out that the toddler had ingested the pesticide, thinking it was milk. He immediately notified the child's parents, and he was rushed to a nearby health facility. At the facility, he was offered milk and activated charcoal (dosage not known) to induce emesis and was subsequently referred.

A referral from the peripheral clinic was made to our facility for further management. The pesticide was identified as Chlorpyrifos (diethyl organophosphate) 50% and Cypermethrin (sodium channel blocker) 5% W/V containing brand used as an acaricide from which 12 - 15ml of pure liquid substance had been ingested.

Objectively, he was fully conscious & irritable, with no other symptoms on arrival at 1100hrs. Thirty minutes later, he became mildly dehydrated, tachycardic at 150 beats per minute, and crying with no other significant symptoms. He was saturating at 96% on room air, breathing at 30 breaths per minute, had a Glasgow Coma Scale of 15/15, and weighed 12kgs. The systematic examination was insignificant to the toddler's presentation.

Student's Journal of Health Research Africa
Vol. 4 No. 9 (2023): September 2023 Issue
https://doi.org/10.51168/sjhrafrica.v4i9.415
Original article

Table 1: Summaryof investigations

Test	Results
CBC:	12.77
WBCs	9.58
GRA	9.6
HBG	451
PLT	
Blood slide for Malaria Parasites	No Malaria Parasites seen
LFTs	4.84
Albumin	.17
Direct Bilirubin	.24
Bilirubin	34
AST	18
ALT	
RFTs	.49
Cr	9.4
Urea	

Pseudocholinesterase was not done due to resource unavailability in our setting.

3. Summary of investigations.

3.1. Management Summary.

Gastric lavage was performed since he was within an hour of ingestion, and intravenous fluids were given to correct dehydration. 12g of activated charcoal was administered using a nasogastric tube while monitoring for the cholinergic crisis. Lavage fluid had a characteristic pesticide smell of petroleum garlic and contained charcoal. Intravenous hydrocortisone was also administered.

At 11:45 am, the intensive care unit was informed to prepare for respiratory support since his GCS dropped to 13/15 with a tachycardic at 160 bpm and 95% oxygen saturation on room air. At 12:30 pm, the pupils became pinpoint with lower limb weakness, and excessive oral and respiratory secretions and atropine administration were done with the starting dose at 0.2mg/kg/dose. The dose was doubled after every 5 minutes until atropinization. Ten minutes later, the child was transferred to ICU for respiratory support with a 74% oxygen saturation on room air, difficulty breathing, and excessive oral pharyngeal secretions.

Oxygen therapy was administered by a non-rebreather mask at 15L/min, together with intravenous pralidoxime. Nebulization with salbutamol and ipratropium bromide was done to relieve the bronchospasms.

Twelve hours later, the toddler improved, with saturations at 97 – 100 %, minimal secretions, and reduced bronchospasm. However, he was febrile with a temperature of 37.7 0C, which subsided with the administration of rectal paracetamol, and he had loose stools.

After 24 hours in intensive care, he was stable and returned to the ward, where his renal and liver function tests were done. He was discharged from the hospital in stable condition and offered a review date of two weeks from the day of discharge.

4. Discussion.

Mixed pesticide poisoning is a significant cause of hospital admission due to the atypical presentation and potentiation of the pesticide's toxic interaction effects. This results from either accidental or intentional poisoning due to the easy availability of these agents as pesticides. Organophosphate, in this case, Chlorpyrifos, results in sig-

nificant toxicity, whereas pyrethroids are considered safe, and their effects are potentiated by the former. The organophosphate is believed to inhibit the enzyme responsible for its inhibition, acetylcholinesterase predisposing to a cholinergic crisis.10 The outcome of this effect is a variety of manifestations, including; Salivation, Lacrimation, Urination, Defecation, Gastric crumps, and Emesis (SLUDGE). Stimulation of nicotinic receptors includes Mydriasis, Tachycardia, weakness, Hypertension, and fasciculations. Muscarinic effects include defecation/diaphoresis, urination, miosis, bronchospasm/bronchorrhea, emesis, lacrimation, and salivation (DUMBELS). These clinical features occur within minutes to hours. Moreover, other patients experience delayed symptoms after an intense period of cholinergic signs and symptoms, minimal symptoms, or none. Other central nervous effects include anxiety, emotional lability, seizures, respiratory and cardiac depression, and extrapyramidal manifestations.11

Organophosphates inhibit the carboxylesterase-mediated metabolism of Cypermethrin, resulting in increased tissue concentration and decreased urine metabolite excretion. Chlorpyrifos Oxon, a toxic metabolite of Chlorpyrifos, irreversibly inhibits the hydrolysis of permethrin, potentiating permethrin toxicity. Cypermethrin is a class II pyrethroid (fig. 1.) with an LD50 of 250mg/kg.12,13 Therefore, the lethal dose for our patient was 3g since he weighed 12kg. However, he had ingested 15ml containing 0.75g, less than the lethal dose.

Similarly, Chlorpyrifos is an organophosphate (fig. 2) with an LD50 of 135mg/kg.12,13 The lethal dose for the 12kg baby translated to 1.62g. However, the patient ingested an equivalent of 7.5g in the 15mls of the substance, which was more than four times the lethal dose. Therefore, the patient was exposed to significant toxicity, necessitating urgent attention. Calculating and establishing the lethal dose is necessary, especially in limited resource settings, since they can guide the decision-making process concerning management.

People exposed to acute pyrethroids exhibit dyspnoea, nausea, headache, and irritability. They also present with excessive salivation and vomiting from excitatory neurotoxicity by disrupting the sodium voltage-sensitive channel.11 Our patient developed non-projectile vomiting and irritability at presentation, later evolving into excessive salivation. The chronic sequelae include cerebral-organic disorders, sensory-motor polyneuropathy involving the lower extremities, and vegetative nervous disorders, such as paroxysmal tachycardia, increased heat sensitivity, and reduced exercise tolerance related to circulatory dysfunction.16 None of these symptoms were present in our patient.

Organophosphate poisoning affects both the sympathetic and parasympathetic. A typical presentation is the latter, except in children, where effects are mainly nicotinic.17 Considering this case, the child had tachycardia and weakness in the lower limbs. Other manifestations were excessive salivation, respiratory secretions, and bronchospasms, which resulted in respiratory distress. Additionally, the patient had miosis due to the muscarinic and nicotinic receptor effects of acetylcholine.

After two weeks from ingestion, organophosphate may also manifest with delayed effects such as intermediate syndrome or organophosphate-induced delayed polyneuropathy (OPIDP).18,19 The patient had not exhibited any delayed toxicity on review two weeks later. However, the assessment may not be reliable owing to the patient's age.

5. Discussion of management.

Management strategies prioritize immediate decontamination with supported pharmacological management. In this case, we asked for the container to be brought to the hospital, which showed an organophosphate poison. Decontamination was done by removing clothes and flushing the patient with tap water. Tefera and Teferi,20 have asserted that personal protection during decontamination must be prioritized to prevent cross-contamination since the poison has easy skin absorption.

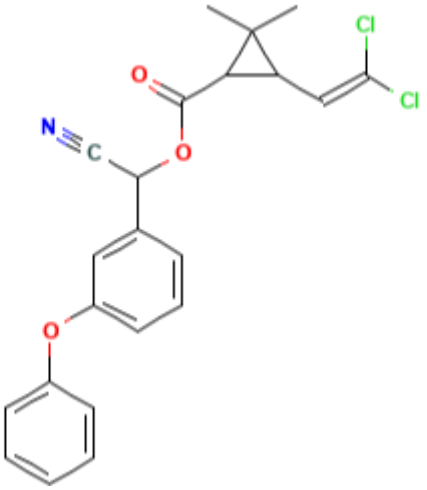

Figure 1: **Cypermethrin Chemical structure.**[14]

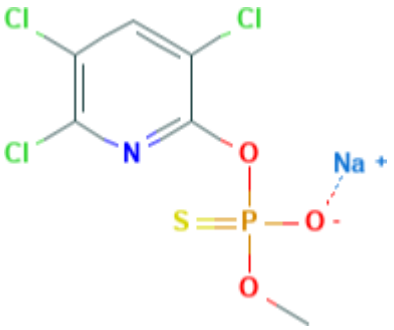

Figure 2: **Chlorpyrifos-methyl-desmethyl sodium.**[15]

Gastric lavage has also been indicated within the first hour of poison ingestion. It decreases absorption by 42% at the 20th minute and 16% at the 60th minute.21 Activated charcoal can be given if the patient presents within 1 hour to reduce poison adsorption.22 Our patient received charcoal from a peripheral clinic which could have lessened the intensity of the presenting symptoms. Airway management must also be prioritized among these patients to maintain respiratory function.23 Intubation has been indicated among patients with bronchospasm and bronchorrhea.24 Additionally, bronchodilators can be administered to the patient. However, succinylcholine should not be used in intubation to avoid prolonged paralysis.19 Different agents have been recommended, for example, anticholinergic agents like atropine and oximes, for example, pralidoxime.18 These are essential in antagonizing the effects of accumulated acetylcholine and promot-

ing the acetylcholinesterase enzyme's action.

Atropine is a definitive treatment by competing with OP at muscarinic receptor starting at a dose of 2 to 5mg IV for adults and 0.02mg to 0.05mg/kg IV among children. Atropine incremental dosing followed by infusion is superior to bolus doses.25

5.1. Adequate atropinization targets.

Day 1 Systolic Blood Pressure > 90mm Hg Heart rate <110, clear lung fields, and the pupils must be mid-position.

Day 2: Heart rate <100

Day 3: Heart rate < 90, with the rest of the days being less than 80.26

Pralidoxime reactivates bound acetylcholinesterase, especially for diethyl but not dimethyl but no benefit in mortality.26 Only benefit is evidence of reactivated acetylcholinesterase. However, if used according to severity APACHE II scores, there was a benefit as demonstrated by Lin et al.27 Abdel-Satar et al.28 has demonstrated effective patient monitoring using pseudocholinesterase levels. However, these may not be possible in limited resource settings, for example, our clinical setting. Pralidoxime is given for the nicotinic effects since atropine works on muscarinic receptors.

The poisoning prognosis highly depends on different factors, for example, duration of exposure to the toxin, quantity of the toxin ingested, immediate patient care, and presence of a sustainable healthcare setting. Additionally, the impact of organophosphate poisoning can be evaluated within the clinical setting depending on toxin identification, body system parameters, and the patient's hemodynamic stability.29 Evaluation of different parameters, for example, pseudocholinesterase, sodium, creatinine, and blood urea nitrogen, is essential in outcome prediction.30 However, few studies have been availed to evaluate the extent of each parameter in prognosis determination among patients following organophosphate ingestion.

5.2. Management of cypermethrin toxicity.

Research has demonstrated little information concerning the availability of an antidote against Cypermethrin.31 However, other literature has demonstrated supportive management as a key intervention in managing individuals exposed to cypermethrin toxins.32 Symptomatic measures among patients exposed to cypermethrin toxins are an augmentation of conservative management. Various measures that have been approved for managing this toxicity include charcoal administration, anticonvulsant medication in case of spasms, adequate hydration and rinsing on skin surfaces, and atropine administration.33,34,35

6. Conclusion.

Organophosphate poisoning remains a public health threat, especially to vulnerable populations, such as children. These may be lured to consume the solutions, thinking it is formula milk. Understanding the immediate presentation is necessary to initiate the appropriate management for the patient. Additionally, early intervention for patients with organophosphate poisoning is fundamental for better outcomes.

7. Recommendation.

Heath regulation bodies must take an interest in educating health workers and the community concerning organophosphate poisoning and its management, especially in a limited resource setting. This will effectively reduce mortality and morbidity.

8. Strength and Limitations.

8.1. Strength.

We involved an interdisciplinary approach in managing this patient as evidenced by consulting the ICU team before the vitals of the patient had deteriorated.

There was close patient monitoring which resulted in better outcomes

8.2. Limitations.

Because of the limited resource setting, we couldn't monitor the pseudocholinesterase assays of the patient. Additionally, toxicology tests were not conducted due to the absence of resources.

9. List of Abbreviations.

ICU	Intensive Care Unit
GCS	Glasgow Comma Scale
ALT	Alanine Transferase
AST	Aspartate Transferase
RFTs	Renal Functional Tests
LFTs	Liver Functional Tests
Cr	Creatinine
GRA	Granulocytes
CBC	Complete Blood Count
WBCs	White Blood Cells
HBG	Haemoglobin
LD	Lethal Dose
WHO	World Health Organization
W/V	Weight per Volume
PLTs	Platelets

10. Acknowledgments.

All authors equally contributed to the case report development.

11. Conflicting interests.

The Authors declare no conflict of interest

12. Funding.

No funding has been received for this case report.

13. References:

1. Robb, E. L., & Baker, M. B. (2017). Organophosphate toxicity. https://europepmc.org/article/NBK/nbk470430
2. Umakanth, M. (2019). Intermediate Syndrome Following Organophosphate Poisoning; Review Article. Asia Pacific Journal of Medical Toxicology, 8(1), 19–24. https://apjmt.mums.ac.ir/article_12401.html
3. Thundiyil, J. G., Stober, J., Besbelli, N., & Pronczuk, J. (2008). Acute pesticide poisoning: a proposed classification tool. Bulletin of the World Health Organization, 86, 205-209.
4. Boyle, K. L., Periyanayagam, U., Babu, K. M., Rice, B. T., & Bisanzo, M. (2020). Pediatric poisonings in a rural Ugandan emergency department. Pediatric emergency care, 36(3), e160-e162. https://journals.lww.com/pec-online/Abstract/2020/03000/Pediatric_Poisonings_in_a_Rural_Ugandan_Emergency.22.aspx
5. Dodson, J., Feng, S. Y., & Rodriguez, D. (2021). "That is Not His Regular Formula": A Case of Organophosphate Poisoning in an Infant. Pediatric emergency care, 37(4), e206-e209. https://journals.lww.com/pec-online/Abstract/2021/04000/_That_s_Not_His_Regular_Formula____A_Case_of.23.aspx
6. Sinha, S. N., Kumar, K. R., Rajendra, S., Gouda, B., Chougule, P., & Naik, R. P. (2022). Nature, risk factors, prevalence, neurotransmitter role, and survival investigation of acute organophosphate (OP) pesticide cases in humans in a tertiary hospital in Telangana, India. https://doi.org/10.21203/rs.3.rs-1544607/v1
7. Odakha, J. A., Harborne, D., & Chen, H. (2022). Predictors of mortality in emergency center patients with acute pesticide poisoning in Uganda. African journal of emergency medicine, 12(3), 242–245. https://doi.org/10.1016/j.afjem.2022.05.005
8. Kwesiga, B., Ario, A. R., Bulage, L., Harris, J., & Zhu, B. P. (2019). Fatal cases associated with eating chapatti contaminated with organophosphate in Tororo District, Eastern Uganda, 2015: Case series. BMC Public Health, 19(1), 1-7. https://link.springer.com/article/10.1186/s12889-019-7143-0
9. Ssemugabo, C., Nalinya, S., Halage, A. A., Neebye, R. M., Musoke, D., & Jørs, E. (2020). Doctor's experiences on the quality of care for pesticide poisoning patients in hospitals in Kampala, Uganda: a quali-

tative exploration using donabedian's model. BMC health services research, 20(1), 1-8. https://bmchealthservres.biomedcentral.com/articles/10.1186/s12913-020-4891-6

10. Adeyinka, A., Muco, E., & Pierre, L. (2018). Organophosphates. https://europepmc.org/article/nbk/nbk499860
11. Uwaifo, F., & John-Ohimai, F. (2020). Dangers of organophosphate pesticide exposure to human health. Matrix Science Medica, 4(2), 27. https://www.matrixscimed.org/article.asp?issn=2521-0807;year=2020;volume=4;issue=2;spage=27;epage=31;aulast=Uwaifo
12. Meenakshi Srinivasan RA, Girish Thunga, Shivashankar Kaniyoor Nagiri, Chandrashekar Udyavara Kudru. Pharmacokinetic Potentiation of Mixed Organophosphate and Pyrethroid Poison Leading to Prolonged Delayed Neuropathy. Journal of Clinical and Diagnostic Research. 2016;10(11):FD01-FD2.
13. Iyyadurai R, Peter JV, Immanuel S, Begum A, Zachariah A, Jasmine S, et al. Organophosphate-pyrethroid combination pesticides may be associated with increased toxicity in human poisoning compared to either pesticide alone. Clinical Toxicology. 2014;52(5):538–41.
14. PubChem. (n.d.). Cypermethrin. Retrieved from: https://pubchem.ncbi.nlm.nih.gov/compound/Cypermethrin
15. PubChem. (n.d.). Chlorpyrifos-methyl-desmethyl sodium. Retrieved from: https://pubchem.ncbi.nlm.nih.gov/compound/154584014
16. Tripathi M, Pandey R, Ambush SP, Pandey M. A Mixture of Organophosphate and Pyrethroid Intoxication Requiring Intensive Care Unit Admission: A Diagnostic Dilemma and Therapeutic Approach. Anesthesia & Analgesia. 2006;103(2):410-2.
17. Yousef, A., Albuali, W., AlOmari, M., AlMutairi, A., Albuali, H. W., AlQurashi, F. O., & Alshaqaq, H. M. (2022). Organophosphate poisoning in a pediatric intensive care unit: a retrospective analysis based on ten years of experience. International journal of general medicine, pp. 15, 6269.
18. Peter JV, Sudarsan T, Moran J. Clinical features of organophosphate poisoning: A review of different classification systems and approaches. Indian Journal of Critical Care Medicine. 2014;18(11):735-45.
19. Robb EL, Baker MB. Organophosphate Toxicity. StatPearls. Treasure Island (FL)2022.
20. Tefera, G. M., & Teferi, L. G. (2020). Prevalence, predictors and treatment outcome of acute poisoning in Western Ethiopia. Open Access Emergency Medicine: OAEM, 12, 365. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7669523/
21. Mugadlimath, A., & Sane, M. (2022). Gastric lavage: savior or superfluous? https://www.indianjournals.com/ijor.aspx?target=ijor:jist&volume=18&issue=1&article=001
22. Chacko, B., & Peter, J. V. (2019). Antidotes in poisoning. Indian journal of critical care medicine: peer-reviewed, official publication of Indian Society of Critical Care Medicine, 23(Suppl 4), S241. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6996653/
23. Eze, J. N., Ndu, I. K., & Edelu, B. O. (2018). Teenage organophosphate insecticide poisoning: An ugly trend in Enugu, Nigeria. Journal of Community Medicine and Primary Health Care, 30(1), 99-108. https://www.ajol.info/index.php/jcmphc/article/view/16862
24. Sivaganabalan, R., Mohd Jamin, Z., Loke, K. Y., Z'aba, N., & Periathamby, S. (2018). Retrospective observational study of organophosphate poisoning in an urban Malaysian Hospital. J Clin Toxicol, 8(2), 1–8.
25. Abedin MJ, Sayeed AA, Basher A, Maude RJ, Hoque G, Faiz MA. An open-label randomized clinical trial of atropine bolus injection versus incremental boluses plus infusion for organophosphate poisoning in Bangladesh. J Med Toxicol. 2012;8(2):108-17.
26. Eddleston M, Eyer P, Worek F, Juszczak E, Alder N, Mohamed F, et al. Prali-

doxime in acute organophosphorus insecticide poisoning–a randomized controlled trial. PLoS Med. 2009;6(6):e1000104.
27. Lin CC, Hung DZ, Chen HY, Hsu KH. The effectiveness of patient-tailored treatment for acute organophosphate poisoning. Biomed J. 2016;39(6):391-9.
28. Abdel-satar, M., Magdi, M., Abdel-ghany, I., & Galal, D. (2022). A Case Study of Neonatal Organophosphate Poisoning Presented as Acute Respiratory Distress. The Egyptian Journal of Forensic Sciences and Applied Toxicology, 22(3), 27-32. https://ejfsat.journals.ekb.eg/article_262060.html
29. Amir, A., Raza, A., Qureshi, T., Mahesar, G. B., Jafferi, S., Haleem, F., & Ali Khan, M. (2020). Organophosphate Poisoning: Demographics, Severity Scores, and Outcomes From National Poisoning Control Centre, Karachi. Cureus, 12(5), e8371. https://doi.org/10.7759/cureus.8371
30. Pannu, A. K., Bhalla, A., Vamshi, V., Upadhyay, M. K., Sharma, N., & Kumar, S. (2022). Changing spectrum of acute poisoning in North India: A hospital-based descriptive study. Turkish Journal of Emergency Medicine, 22(4), 192199.
31. Pannu, A. K., Bhalla, A., Vishnu, R. I., Garg, S., Dhibar, D. P., Sharma, N., & Vijayvergiya, R. (2021). Cardiac injury in organophosphate poisoning after acute ingestion. Toxicology Research, 10(3), 446–452. https://doi.org/10.1093/toxres/tfab036
32. Seven, B., Yalçin, E., & Acar, A. (2022). Investigation of cypermethrin toxicity in Swiss albino mice with physiological, genetic, and biochemical approaches. Scientific Reports, 12(1), 1-15. https://www.nature.com/articles/s41598-022-15800-8
33. Ranjan, A., & Jindal, T. (2022). Management of Human Organophosphate Poisoning. In Toxicology of Organophosphate Poisoning (pp. 91-99). Springer, Cham. https://link.springer.com/chapter/10.1007/978-3-030-79128-5_6
34. Chang, Y. C., Ge, X., Wang, L. J., Lee, S. S., Paulsen, M. H., Khan, Q. M., ... & Lin, Y. (2018). An ultra-low-cost smartphone device for in-situ monitoring of acute organophosphorus poisoning for agricultural workers. Sensors and Actuators B: Chemical, 275, 300-305. https://doi.org/10.1016/j.snb.2018.08.009
35. Joko, T., Dewanti, N. A., & Dangiran, H. L. (2020). Pesticide poisoning and the use of personal protective equipment (PPE) in Indonesian farmers. Journal of environmental and public health, 2020. https://doi.org/10.1155/2020/5379619

14. Publisher details:

Publisher: Student's Journal of Health Research (SJHR)
(ISSN 2709-9997) Online
Category: Non-Governmental & Non-profit Organization
Email: studentsjournal2020@gmail.com
WhatsApp: +256775434261
Location: Wisdom Centre, P.O.BOX. 148, Uganda, East Africa.