Organophosphate poisoning from inappropriate topical use of malathion pesticide: A case report

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Abstract
A 43-year-old male with no past medical history presented to our emergency department with vomiting, diarrhea, and abdominal pain of 3 h’ duration. Upon further questioning, he revealed that he had been applying malathion pesticide over his body for the past 3 days for self-diagnosed scabies. He was otherwise afebrile and hemodynamically stable, and the physical examination was unremarkable. The patient was diagnosed with organophosphate poisoning and treated symptomatically due to the lack of worrying cardiorespiratory or neurologic sequelae. He was subsequently admitted to the general ward, where his symptoms abated within 4 h. Serum and red blood cell cholinesterase tests sent on admission returned on day three and were significantly decreased (serum cholinesterase 2131 U/L, reference range 4700–12000 U/L; red blood cell cholinesterase 3365 U/L, reference range 7700–14600 U/L). He was discharged home well and stable on day 5 of admission, with outpatient psychiatric follow-up for likely delusional parasitosis.

Keywords
Malathion, organophosphate poisoning, pesticide

Introduction
Malathion is an organophosphate compound used in pharmaceutical formulations for the treatment of scabies. In the agricultural setting, it is a potent pesticide and has the potential to cause systemic toxicity when ingested orally. We present a novel case of organophosphate poisoning in an adult from repeated topical exposure.

Case report
A 43-year-old male with no past medical history of note presented to our emergency department with acute onset nausea, vomiting, and diarrhea of 3 h’ duration, associated with generalized abdominal discomfort. He denied any dietary indiscretion, unwell contacts, fever, chest pain, or breathlessness.

Upon further questioning, the patient revealed that he had been applying malathion pesticide (500 g/L malathion in 487 g/L liquid hydrocarbon solvent; Figure 1) mixed with lotion thrice a day for a self-diagnosed scabies infection over the past 3 days. He last applied malathion 3 hours before presentation and immediately showered thoroughly after onset of symptoms. He denied ingestion of malathion at any point.

Upon presentation, the patient was afebrile and hemodynamically stable, with a heart rate of 85 bpm and blood pressure of 161/92 mmHg. On examination, he was alert and responding relevantly to questioning. Pupils were 2 mm in diameter, equal, and reactive to light. There was no excessive lacrimation or salivation noted. His chest was clear to auscultation, with no crepitations or rhonchi. His abdomen was soft and non-tender to palpation with no signs of peritonism. Neurological examination did not reveal any fasciculations or paralysis. Multiple excoriations were noted on skin examination, but no scabetic tracks or burrows were seen. Serum and red blood cell cholinesterase levels were sent to the laboratory for testing. A chest X-ray was performed, which did not show any opacities or airspace consolidation.
After discussion with toxicology, the patient was subsequently started on symptomatic treatment with anti-emetics. Atropine and pralidoxime were withheld in view of the patient’s mild presentation, with primarily gastrointestinal symptoms and lack of cardiorespiratory or neurological toxicity. The patient was admitted to the general ward from the emergency department, and his symptoms of vomiting and diarrhea resolved with symptomatic treatment within 12 h. Skin scraping done was negative for the scabies mite *Sarcoptes scabiei*.

Over the next 3 days, the patient was evaluated by the inpatient psychiatric team, and was eventually diagnosed with delusional parasitosis after organic causes were ruled out. The serum and red blood cell cholinesterase tests sent on admission returned on day three of admission and were significantly decreased (serum cholinesterase 2131 U/L, reference range 4700–12000 U/L; red blood cell cholinesterase 3365 U/L, reference range 7700–14600 U/L). The patient was discharged well on day five of admission with outpatient psychiatric follow-up.

**Discussion**

Malathion, along with other organophosphates and closely related carbamates, is an anticholinesterase compound primarily used in agriculture for their pesticidal properties. These potentially toxic compounds are unfortunately the leading cause of pesticide poisoning worldwide and kill approximately 100,000 people each year, primarily in developing countries.1,2

Organophosphates can be absorbed systemically via all routes, namely, ingestion, inhalation, and contact. They exert their toxic effects via inhibition of acetylcholinesterase, leading to increased acetylcholine levels at cholinergic synapses.3 Muscarinic hyperstimulation clinically manifests as the “SLUDGE” symptoms (salivation, lacrimation, urination, diaphoresis, gastrointestinal upset, and emesis), whereas nicotinic hyperstimulation manifests as tachycardia, hypertension, mydriasis, weakness, and muscle fasciculations.4

The diagnosis of organophosphate poisoning is usually clinically apparent from the history or presenting toxidrome. In unclear circumstances, serum and red blood cell cholinesterase levels can be sent for laboratory testing; since these levels are significantly lowered once the patient is symptomatic, normal cholinesterase levels excludes systemic organophosphate poisoning.5 In this case, as the differential diagnosis included acute infective gastroenteritis and we were not aware of reported clinically significant cases caused by dermal absorption of malathion, cholinesterase levels were sent to confirm the diagnosis.

The initial treatment of organophosphate poisoning, as with all toxic exposures, consists of source control and patient decontamination. No further decontamination was attempted for this patient as he had already done so prior to presentation. Two broad classes of antidotes are available for organophosphate poisoning. Antimuscarinic agents (e.g., atropine and glycopyrrolate) have rapid onset and are indicated when muscarinic effects are severe or life-threatening. Another treatment consideration is the administration of oximes (e.g., pralidoxime), which regenerates acetylcholinesterase and improves both muscarinic and nicotinic effects. However, the clinical evidence behind oxime therapy is lacking, with a recent Cochrane review6 concluding that the current evidence is insufficient to indicate clinical benefit or harm with oxime therapy. In this case, due to the lack of “killer B” symptoms (i.e., bradycardia, bronchorrhea, and bronchospasm) indicating severe muscarinic hyperstimulation, the decision was made to withhold atropine and pralidoxime.

Pure malathion has a low toxicity and a low propensity for dermal absorption,2 especially when compared to other organophosphate pesticides (e.g., chlorpyrifos and diazinon).7 Under controlled conditions in in vivo settings, malathion was noted to be less efficiently absorbed after dermal exposure than after oral exposure,8 with two volunteer studies showing approximately 7% absorption when malathion in ethanol solution was applied topically.8,9 However, an in vitro skin flap study found that although systemic absorption was 7–9% of the dose applied, up to 34% of the dose applied...
was present in the skin and stratum corneum and represented a source of potential continued absorption.10

To our knowledge, this is the first reported case of systemic organophosphate toxicity in an adult from purely cutaneous malathion exposure, for an apparently therapeutic indication. Previous cases of systemic toxicity from mixed but primarily cutaneous exposure have been reported in children,11,12 who are more susceptible due to their higher surface area to body weight ratio.13

We postulate three possible factors that could have led to significant dermal absorption in this case. First, the use of organic hydrocarbon solvent in malathion pesticide differs from aqueous solvents used in malathion for therapeutic use, which may lead to increased absorption.13 Changing solvents is known to increase toxicity of other organophosphate pesticides.14 Second, his natural skin barrier could have been disrupted by scratching; the presence of broken skin is known to enhance cutaneous absorption of organophosphates.2,15 Last, his repeated applications over 3 days could have created a skin reservoir of malathion for continued absorption.

Conclusion

We present this case as a novel clinical example of malathion toxicity from dermal absorption through a misuse of pesticidal product for pharmacological use. More inquiry is needed to determine the safety and toxicity of topical human exposure to malathion pesticide, especially in cases of acute repeated exposure.

Author contributions

JC wrote the first draft of the manuscript. All authors reviewed and edited the manuscript and approved the final version of the manuscript.

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Availability of data

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

Ethical approval

(include full name of committee approving the research if available mention reference number of that approval)

Changi General Hospital does not require ethical approval for reporting individual cases or case series when informed consent is obtained and all identifiable patient data is anonymized.

Informed Consent

Written informed consent was obtained from the patient(s) for their anonymized information to be published in this article.

Trial Registration

(where applicable)

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