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Letter to the Editor

COMMENTS ON "A SYSTEMATIC REVIEW OF ALUMINIUM PHOSPHIDE POISONING"

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We have read with curiosity the systematic review of aluminium phosphide poisoning by Mehrpour et al. (1) recently published in the *Archives of Industrial Hygiene and Toxicology*. They mentioned potassium permanganate (1:10,000 solution) as an option in the management of aluminium phosphide toxicity and claimed that it can oxidise phosphine gas in the stomach to phosphate, and therefore decrease the quantity of lethal phosphine gas. Here the authors cite two case reports using potassium permanganate (1:10,000 solution) without any strong scientific basis (2, 3).

As we know, in contact with mucus potassium permanganate ($KMnO_4$) forms manganese dioxide, potassium hydroxide, and free oxygen radical. Therefore it can lead to systemic toxicity itself (4). Because of its weak bonds with the aluminium-based compound, phosphine gas (PH_3) can easily be released after ingestion (5). PH_3 is a hard nucleophile (species with increased electron density over a small area) and therefore tends to react with hard electrophiles (4, 6), but phosphine, the small nucleophile, and free oxygen radical, another small nucleophile, do not interact with each other.

The only clinical indication for potassium permanganate prescription may be white phosphorus (P_4) ingestion, as it converts it to a less destructive oxide compound. However, due to its chemical properties, clinicians should be aware of the risk of systemic toxicity. In addition, no clinical trials have yet determined its risk-to-benefit profile (4).

We think that prescribing potassium permanganate in aluminium phosphide poisoning stems from an ancient misperception of the chemical structure of phosphine (PH_3) *versus* white phosphorus (P_4).

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