A Suicide Attempt Using Zinc Phosphide
(A Case Study)

Zinc Phosphide ile Meydana Gelen Bir Suisid Vakası
(Olgu Sunumu)

Zinc Phosphide ile Suisid Girişimi / Suicide Attempt Using Zinc Phosphate

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Özet
Çinko Fosfürü, fare mücadelesinde zehirli buğday üretimi için kullanılır ve rodentinin aktif maddesidir. İntihar amacıyla tarım ilaçları [Zinc Phosphide (Zn3P2)] içen 17 yaşındaki erkek olgu acil serviste görüldüğünde: Genel durumu kötü, bilinci kapalı, kusmuğu ve cildinde sarımsak kokusu ve ileri derece asidozu mevcuttu. Semptomatik tedavi uygulanan ve mekanik ventilatorda takip edilen olgu yatışının 5. günü psikiyatri kliniğine devirildi.

Anahtar Kelimeler
Çinko Fosfürü; Suisid; Sarımsak Kokusu

Abstract
Zinc phosphide is a toxin that is added to wheat for use in rodent control and is the active ingredient of rodenticide. A 17 year-old male attempted suicide by drinking pesticide [Zinc PHOSPHIDE (Zn3P2)] and was subsequently admitted to the emergency department: the patient’s general condition was poor, he was unconscious and vomiting, the skin had a garlic odor and advanced acidosis was present. The patient was treated symptomatically, followed by mechanical ventilation, and was transferred to a psychiatric clinic on the fifth day.

Keywords
Zinc Phosphide; Suicide; Smell of Garlic

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Introduction
Organophosphate poisoning is a significant cause of morbidity and mortality in small- and economically active age groups from developing countries all over the world, particularly those of low educational and socio-economic status. Zinc phosphide, the active material of rodenticide, is used in the production of toxic hydrogen gas (PH₃, phosphine) emerging after dissolution upon meeting a gas. Both history of exposition and the smell of garlic or rotten fish help diagnosis. The lethal dose of zinc phosphide is 20-40 mg/kg for humans [1].

We present the case of a 17 year-old male who attempted suicide by drinking pesticide [Zinc Phosphide (Zn3P2)].

Case Report
When the patient was observed in the emergency department; general condition was poor, he was unconscious, intubated, was unresponsive to painful stimulus, in GCS-3, papillary was advanced miotic (pin point), light response was +/-, respiratory sound was coarse with increased secretion. Relatives of patients given the drug by the time and dose was unclear zinc phosphide. The case was vomiting and had a garlic smell from his skin, and was isolated for 48 hours to prevent the poisoning of other patients. His noninvasive blood pressure was 80/40 mmHg, and pulse was 94/min. Toxicological information sources were consulted. The patient was admitted to the intensive care unit to prevent the progression of metabolic acidosis, hypoxia, hypoglycemia, ventricular dysrhythmia, hypotension, tachycardia, ataxia, hepatotoxicity effect, acute renal failure. Analysis of arterial blood gas showed that pH was 7.11, PaO2 as 44 mmHg, HCO₃ as 14.6 mmol/L and SpO2 as 85%. Propofol infusion was commenced and followed as FiO2:0.5, Vt:6 mL/kg, frequency:14/minutes, and PEEP:5 cmH2O in SIMV mode in a mechanical ventilator. The patient was given Zmg/kg activated carbon (Char-Flx) with a naso-gastric catheter. 2000cc fluid per day was given to the patient, urine output was observed as normal. Liver function tests and other biochemical parameters remained normal values. Hemodynamics remained stable. Additional drug treatment did not implemented. The case did not require atropine. The patient’s papillary healed after 6 hours. Further analysis of blood gas showed that pH:7.38, PaO2:87 mmHg, PaCO2:34mmHg, HCO3:21 mmol/L, SpO2:95%. Morning sedation was ceased and the patient was extubated on the second day. Additive pathology was not detected in tracks. The patient was transferred to a psychiatry clinic on the fifth day when he did not require further intensive care.

Discussion
In the mechanism of zinc phosphide poisoning; poisoning is mediated by phosphine, which was thought to act by inhibiting cytochrome C oxidase. While phosphine does inhibit cytochrome C oxidase in vitro, the inhibition is much less in vivo. It was recently shown in nematodes that phosphine rapidly per - tubular acidosis (type 1) and deep metabolic acidosis (ph:6.973, PaCO2:24.2 mm Hg, PaO2:91 mm Hg, and HCO3:9.3 mEq/L, lactate: 2 mEq/L) in a 25 year-old zinc phosphide poisoned male patient. They recommended that respiratory support and intravenous fluid resuscitation with sodium bicarbonate treatment should be considered.

Vomiting should be avoided, as patients develop toxic phosphine when he did not require further intensive care.

Competing interests
The authors declare that they have no competing interests.

References