

**CASE REPORT****TRANSIENT LEUCOPENIA IN ACUTE ZINC PHOSPHIDE POISONING**Niraj Keyal¹, Hem Raj Paneru¹, Gentle Sunder Shrestha¹, Roshana Amatya¹¹ Department of Anaesthesiology, Tribhuvan University Teaching Hospital, Maharajgunj, Kathmandu, Nepal

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ABSTRACT

Zinc phosphide is easily available, cheap, and highly effective household rodenticide. Self poisoning with this rodenticide is commonly encountered in our clinical practice and causes lethal poisoning by liberation of phosphine which in turn causes inhibition of cytochrome oxidase C thereby leading to cellular hypoxia. We report a case of such poisoning which had an unusual presentation in the form of transient leucopenia.

Key words: Rodenticide, Transient leucopenia, Zinc phosphide.

INTRODUCTION:

Zinc phosphide is easily available, cheap, and highly effective household rodenticide available in the form of black powder containing 75% of Zinc Phosphide and 25% of Potassium Tartarate.^[1] Poisoning with this compound is possible by accidental exposure or intentional self poisoning. When ingested it transforms into phosphine gas which is rapidly absorbed from the gastrointestinal mucosa and irreversibly inhibits Cytochrome oxidase C thereby causing tissue hypoxia. Till date, there is no antidote for this poisoning and the mortality rate is very high (37 – 100%).^[2]

CASE HISTORY:

Forty-one years old male, presented to emergency department with history of self poisoning with 2 grams of 3 months old, seal packed Zinc phosphide with consumption of few sips of water following ingestion of the poison followed by few episodes of vomiting. He had no significant past medical or surgical history except for regular alcohol consumption once a week.

Patient presented to emergency room one hour

after ingestion of poison and was conscious, well oriented to time, place, and person, with Glasgow Coma Scale (GCS) of 15/15. His pulse rate was 90 beats per minute, blood pressure 110/60 mmHg, respiratory rate 16 breaths per minute, oxygen saturation of 95% on room air, and temperature 98.5°F. Respiratory, cardiovascular and per abdominal examination findings were normal. All the routine lab parameters including chest x-ray, 12 lead Electrocardiogram (ECG), Arterial blood gas renal function, liver function, urine routine and microscopic examination, blood sugar were within normal limits with hemoglobin of 10 gm%, White Blood Cell (WBC) count of 6000/mm³, neutrophils 80%, lymphocytes 15%, and platelet count of 180,000/mm³.

He underwent gastric lavage with Normal saline in the emergency department within 15 minutes of arrival and was admitted to intensive care unit for further monitoring and management. On the second day of admission he developed sore throat with difficulty in swallowing. His WBC counts decreased to 3380/mm³ with neutrophils 80%, and

lymphocytes 15% with hemoglobin of 10 gm% and platelet counts of 181,000/mm³. Complete blood counts were ordered daily thereafter which revealed total counts of 2200/mm³ with neutrophils 70% on day three, 3000/mm³ with neutrophils 72% on day four which progressively increased and reached 6000/mm³ with neutrophils 74% on day seven.

He never developed fever, tachycardia, tachypnea, absolute neutrophil count(ANC)<500/mm³during ICU stay, nausea and vomiting also improved and was discharged home after 8 days of hospital stay. He did not develop any complications during the follow up period of 2 weeks.

DISCUSSION:

Zinc phosphide leads to death in massive doses (more than 4-5 grams). In a study, the average age of patients who attempted suicide was reported to be 27 years.^[3]

There are many reports that higher doses of this drug (4-5 grams) may rapidly lead to death but sometimes doses more than 5 grams may only induce vomiting and some other clinical manifestations.^[4]

Chronic and excessive Zinc intakes have been associated with hematological problems including leucopenia. The mechanism appears to be zinc-induced copper deficiency, which is instrumental in producing the profound bone marrow abnormalities, as zinc itself is of low toxicity.^[5,6,7]

Literature on hematological effects of acute Zinc Phosphide poisoning is scarce. In a case report, Ali et al have reported unexplained transient leucopenia in Zinc Phosphide poisoning.^[8]

We did not measure serum copper level though hypocupremia is suggested to be possible underlying mechanism leading to hematological toxicities, the effect of acute Zinc ingestion in the form of Zinc phosphide is not clear yet.^[9]

Our patient did not have any medical illnesses, congenital or acquired, which could explain the onset of leucopenia. He did not have any clinical symptoms and signs of infection. Biochemical markers for infection and cultures were all negative. All the laboratory investigations in the line leucopenia revealed normal findings.

CONCLUSION:

To conclude, leukopenia can be an acute, transient, self limiting effect of Zinc Phosphide poisoning that does not require treatment. It should be looked for in all Zinc Phosphide poisoning cases. Mechanism of action to cause leucopenia is not clear in acute Zinc Phosphide poisoning and large scale studies are required to understand the mechanism of action and course of poison.

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