CASE REPORT

Pretilachlor Poisoning: A Rare Case of Herbicide Poisoning with Neurotoxicity

Shaik Khasim1*, Sri Karan Uddesh Tanugula2, Kasturi Ravinder Reddy3

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Abstract

Pretilachlor poisoning in humans is an understudied area. It is widely used as a herbicide in India; hence it is of paramount importance that we understand the various clinical presentations from its toxicity. Acute oral intoxication of pretilachlor can present with neurological and gastrointestinal manifestations. This case report adds to the scant evidence on how to recognize and manage pretilachlor toxicity.

Introduction

Pretilachlor is a synthetic chloroacetanilide herbicide. Pretilachlor is a broad-spectrum systemic herbicide with the chemical name 2-chloro-2'-6'-diethyl-N-(2-prop-oxyethyl) acetanilide. The mechanism of action of this group of herbicides is still not clearly understood but is known to act by inhibiting the biosynthesis of fatty acids, lipids, proteins, and flavonoids.

We report a case of a 58-year-old male who presented with neurological and gastrointestinal manifestations following oral ingestion of pretilachlor.

Case Description

A 58-year-old Indian male presented to the emergency department of Prathima Institute of Medical Sciences, Karimnagar with an alleged history of oral ingestion of 150 mL of undiluted pretilachlor (the said chemicals empty bottle was brought along with the attenders) following which the patient developed burning sensation in throat and two episodes of vomittings. The patient was initially taken to a local hospital within 1 hour, where gastric lavage was done with normal saline, and then shifted to our hospital for further management. On admission the patient was conscious and his initial vital parameters were as follows: blood pressure was 130/90 mm Hg; pulse rate was 105 beats per minute, respiratory rate 22 breaths per minute, SpO2 95% on room air, capillary blood glucose level 130 mg/dL. There was no history of chest pain, palpitations, fever, seizure, or loss of consciousness.

On the first day of hospitalization, patient had multiple episodes of vomitings and burning sensation in throat for which he was managed conservatively with antiemetics and proton pump inhibitors. After 12 hours of hospitalization, patient reported headache and giddiness. His sensorium worsened and patient had two episodes of generalized tonic-clonic seizures, each lasting for about 2–3 minutes. His vitals at that moment were as follows: blood pressure was 80/50 mm Hg, pulse rate was 52 beats per minute, respiratory rate was 29 breaths per minute, SpO2 90% on room air, capillary blood glucose level 210 mg/dL, and had plenty of oral secretions. He was given benzodiazepines and anti-seizure medication (levetiracetam).

On neurological examination, plantar reflexes were absent, adequate bilateral pupillary response was noted, and Glasgow Coma Scale (GCS) score was 5/15 (eye: 1, verbal: 2, and motor: 2). In view of poor GCS the patient was intubated, trachea was secured, and connected to mechanical ventilator. He was immediately started on intravenous (IV) fluids, antiemetics, IV benzodiazepines, and anti-seizure medication. The patient developed hypotension for which fluid challenge with normal saline was given, despite the fluid challenge the blood pressure did not improve; hence the patient was started on inotropic support.

Magnetic resonance imaging of the brain was normal. Serum sodium, potassium, and chloride were within normal limits. Serum magnesium, serum phosphorous, and serum calcium were done and they were normal. Complete blood picture (CBP) showed neutrophilia. Liver function test (LFT) revealed hypoalbuminemia (2.5 gm/dL). Creatinine and blood urea nitrogen were normal. Chest X-ray was normal. Electroencephalogram findings revealed diffuse cerebral dysfunction with generalized seizure activity.

Over the next 2 days of hospitalization, supportive therapy was given as stated above, along with the addition of IV human albumin, regular chest physiotherapy, and endotracheal tube care. Daily monitoring of CBP, LFT, and creatinine was done. Over the next 48 hours, the patient’s clinical status improved. He was weaned off from mechanical ventilation and inotropic support was tapered and stopped. Over the next 2 days of hospitalization, his clinical parameters improved significantly. He was discharged from the hospital on the 7th day after behavioral counseling. After a week the patient came for follow-up visit and he was doing well.

Discussion

Pretilachlor is a synthetic chloroacetanilide. Chronic exposure to chloroacetanilide in vitro and in vivo studies has shown that it has a role in causing neurotoxicity, genotoxicity, and carcinogenicity. The literature has minimal data on how to treat chloroacetanilide poisoning. The cause of the central nervous system (CNS) manifestations might be the direct effect of pretilachlor or the solvents added in the herbicides. Central nervous system manifestations portend a grave prognosis, hence clinicians must be aware of the protean manifestations of its toxicity; this awareness would lead to earlier diagnosis and better-targeted treatment.

One study by Lo et al. in 113 patients with oral exposure to chloroacetanilides suggested that around 25% of the patients were asymptomatic, the rest had gastrointestinal manifestations like vomiting, gastritis, and CNS manifestations like seizures, coma, and stupor were reported along with three

1Resident; 2Consultant Physician, Department of General Medicine; 3Professor and Head, Department of Medicine; Consultant Cardiologist, Prathima Institute of Medical Sciences, Nagunur, Telangana, India; *Corresponding Author

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fatalities which were due to circulatory collapse.\textsuperscript{5} A retrospective study of 35 patients with acute oral chloroacetanilide poisoning concluded that it was found to be of low toxicity in most patients while three patients were comatose and one patient died 24 hours after the exposure.\textsuperscript{4}

There is no antidote available as of today hence the mainstay of treatment is symptomatic with initial stabilization of the patient, decontamination, IV fluid resuscitation, and close hemodynamic monitoring.\textsuperscript{6} In our case, we managed the patient symptomatically with IV fluids, antiemetics, antiepileptics, inotropic support, and ventilator support.

**CONCLUSION**

In conclusion, pretilachlor poisoning causes neurotoxic symptoms which range from giddiness, seizures to coma. Education and awareness among the treating physician regarding the neurotoxicity of pretilachlor is an important aspect that needs to be noted from this case report. Initial stabilization, close monitoring, and supportive treatment are the mainstay of management for pretilachlor poisoning.

**REFERENCES**