CASE REPORT

The unexpected impersonator: A rare case of pretilachlor herbicide poisoning

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Abstract

Pretilachlor is an extensively used herbicide worldwide that mimics the clinical presentation of Organophosphorus (OP) poisoning upon ingestion. Misdiagnosis can be a major setback and lead to inappropriate patient management. In this report, we discuss the case of a young man who presented with an alleged history of consumption of an unknown chemical compound, which was later confirmed to be pretilachlor. He had multiple episodes of emesis since the event, and the only systemic abnormality was a low heart rate of approximately 50 beats/min. Other cholinergic symptoms were absent, and atropine was administered after initial resuscitation and stabilization, with suspected OP poisoning as the top differential diagnosis. All laboratory findings, including pseudocholinesterase, were normal, which was unlikely to happen in the case of OP poisoning. Later, the consumption of the chemical pretilachlor, by the patient was confirmed. The patient was weaned off atropine and managed by systemic support and symptomatic treatment. He fully recovered within 4–5 days and was discharged in a stable condition with no risk of post-discharge complications.

Keywords: Pretilachlor, Organophosphorus Poisoning, Chemical Toxicology

Introduction

Pretilachlor, belonging to the class of chloro-acetanilides, generally marketed in a 50% emulsifiable concentrate formulation, is a group of extensively used herbicides across the world with various names, including Alachlor, Etachlor, Propachlor, etc. It is synthesized in a stepwise manner using chemicals like sodium propanolate and other solvents. There is no naturally occurring plant-based source for this herbicide. It is selective with a broad-spectrum usage as a pre-emergence herbicide for transplanted rice, applied within 4 days of transplanting crops and controls the growth of almost all weeds (annual grasses, edges, and broad-leaved weeds). It is highly economical owing to its broad-spectrum activity and ease of use in providing long-duration control. Other crops that benefit from these herbicides include corn, soybean, and sorghum. Many states in India see the widespread usage of this manufactured chemical owing to extensive agricultural practices [1]. It is estimated that 90% of fatal pesticide poisoning occurs in developing countries [2]. The basic mode of action has yet to be completely understood; however, it is known that this class of herbicide inhibits the biosynthesis of lipids, alcohols, fatty acids, proteins, isoprenoids, and flavonoids [1]. Acute pretilachlor intoxication via ingestion can be easily mistaken for more commonly seen pesticide poisoning, such as organophosphates, resulting in inappropriate management of the patient and futile utilization of resources not required in such cases. Chronic exposure to these herbicides has shown to have probable carcino-genic effects [3]. In this report, we discuss a similar case encountered at a tertiary care hospital in South India.

Case Report

A 32-year-old male patient without any relevant medical history presented to the emergency room with an alleged history of consumption of an unknown poison in the morning, following a family dispute over finances. The patient mentioned that the quantity was approximately 100 ml; however, he was unsure about the exact name or nature of the substance. He immediately experienced 10–12 episodes of vomiting (non-bilious, non-projectile) following the ingestion, mostly containing food particles. There were no other associated symptoms on presentation. On initial examination, the patient was conscious, oriented, and afebrile, with a GCS score of 15/15. Except for vomiting, the patient showed no other cholinergic symptoms. He had a low heart rate of 50 beats/min with other vital parameters being stable and blood oxygen saturation (SpO_2) of 100% on room air. Systemic examination revealed a clear chest and a soft and non-tender abdomen.

A provisional diagnosis of Organophosphorus Poisoning (OP) was made, and intravenous atropine was immediately initiated. Following the administration of 0.6 mg bolus dose of intravenous atropine, the heart rate transiently increased to 110 beats per minute and then went down to 60 beats per minute. A 12-lead electrocardiogram showed a normal sinus rhythm.

Gastric lavage and dermal decontamination were performed. Skin cleaning was performed thoroughly using soap and water and treatment was initiated with 2 ampoules (0.6 mg each) of intravenous atropine, administered 8^{th} hourly with continuous monitoring of vitals and ongoing supportive management. Intravenous fluids and antiemetic were administered.

Pseudocholinesterase levels were checked on two occasions one day after admission and remained within normal limits, showing no significant decline (4775 U/L and 4077 U/L respectively). Complete blood count, and renal and liver function tests did not reveal any abnormalities. Toxicology screening was conducted, and a blood panel with a thin-layer chromatography sample tested positive for a compound consistent with OP. The stomach content pesticide panel also yielded the same result; however, the urine toxicology panel was negative for compounds consistent with organophosphorus. After continuing to remain admitted in the intensive care unit for observation, the patient's family brought to us an opened unlabeled bottle they found in the corner of his bathroom. The herbicide content was confirmed to be pretilachlor after chemical screening using conventional methods for pesticide residue and metabolite detection with gas chromatography and high-performance liquid chromatography. The patient was then weaned off atropine and symptomatic treatment was continued. Since this was the first suicide attempt triggered by unknown conditions, a psychiatry and clinical psychology consultation was sought. Management with antidepressants was recommended to prevent similar attempts at life in the future. The need for follow-up was also explained to the patient and his family. He was administered clonazepam (0.5 mg) and escitalopram (10 mg) tablets once daily until further evaluation. The patient was closely monitored, and after an uneventful period of hospitalization for 4-5 days, he was discharged and asked to follow up for two weeks in the outpatient department

Discussion

Pretilachlor is a colorless and odorless liquid herbicide that can mimic OP poisoning if ingested for the purpose of self-harm. This can lead to misdiagnosis by the treating physician; hence, utmost clarity must be sought to prevent such a situation. Accidental exposure to chloroacetanilides can occur via several routes, including in the diet as residues on treated raw agricultural commodities, and via drinking water as residues found in groundwater that can be used as water sources [4]. Rarely, individuals may ingest this herbicide with the intent of self-harm. Accidental ingestion of this chemical has also been noted to occur in the past. This herbicide is known to produce cholinergic symptoms like OP; however, does not require treatment with atropine.

As seen from the reviewed literature, all patients with pretilachlor poisoning survived without complications and were mostly asymptomatic at the initial presentation (Table 1). Symptomatic treatment with stabilization is the gold standard for treating such patients. Incorrect diagnosis and confusion with OP poisoning may lead to inappropriate and unnecessary drug use and prolonged treatment time.

Another significant finding, as seen in our case, is the importance of retrieving the poison container whenever possible to identify the active compound used. Redesigning containers with precautionary warning labels for the public and restricting the availability of this herbicide over the counter is highly recommended [3].

More effective means of diagnosing the exact toxin would also reduce the time consumption and prevent delays in correct management. Chronic exposure to chloroacetanilide causes neurotoxicity, genotoxicity, and carcinogenicity *in vitro* and *in vivo* [3].

A retrospective study of 35 patients with acute oral chloroacetanilide poisoning concluded that although it was found to have low toxicity in most of the patients, three patients were comatose, and

Table 1: List of previously reported pretilachlor poisoning cases

Authors	Brief about case/(s)	Patient outcome
Dewan [5] (2017)	14 cases of pretilachlor poisoning	All patients survived
Prashar and Ramesh [6] (2018)	4 cases of pretilachlor poisoning	
Parekh and Gupta [7] (2019)	2 cases with the alleged history of poisoning with pretilachlor	
Sarkar and Santra [8] (2022)	10 cases of pretilachlor poisoning over 1 year period, 50% being asymptomatic, 3% with vomiting and throat discomfort, 1% with oral ulcerations and decreased urination	
Khasim *et al*. [9] (2022)	1 case of pretilachlor poisoning with neurotoxicity and gastro-intestinal symptoms	

one patient died 24 h after the exposure. Another study by Lo *et al*. [10] in 113 patients with oral exposure to chloroacetanilides such as alachlor and butachlor suggested that approximately one-fourth of the patients were asymptomatic, while the rest had vomiting and neurological symptoms ranging from drowsiness to central nervous system depression and three fatalities after manifesting profound hypotension and coma. In this case, the patient presented with bradycardia, vomiting, and no other symptoms, which led to the initial diagnosis and treatment of OP poisoning. The absence of typical muscarinic and nicotinic dysfunction as seen in OP poisoning, such as urinary incontinence, excessive lacrimation, salivation, diarrhea, miosis, or the distinct characteristic garlicky odor, were other clues that pointed towards an alternate diagnosis. The point of confusion was incomplete information about the exact nature of the chemical taken by our patient, and a provisional diagnosis of OP poisoning was made because of excessive vomiting and bradycardia.

Another point to highlight in this case was the absence of complications and the lack of need for follow-up and monitoring post-discharge, except for psychiatric evaluation. In cases of OP poisoning, post-treatment complications, such as intermediate syndrome or OP-induced delayed polyneuropathy, should be strictly monitored. Pretilachlor poisoning has a 100% survival rate, as the management consists of only symptomatic treatment and monitoring without the need for aggressive measures or specific antidotes. There were also no residual adverse effects in the patient after completion of the treatment link while there is a continued risk of health dysfunction or organ damage in OP poisoning. Pseudocholinesterase laboratory values are also markedly low in the case of OP poisoning, which was not observed in our patient. Hence, these values can be good indicators to confirm the diagnosis and must be tested early to avoid confusion.

Despite being the first case of intentional pretilachlor ingestion encountered by a group of physicians in this hospital, it must be emphasized that this is not an uncommon occurrence. Due to the easy availability of this compound in our city and because most of the population is involved in agriculture, it is easy for individuals to have easy access. Social stressors which led to our patient drinking this herbicide to end his life, highlight the importance of psychiatric counselling and reassurance to help such individuals. Proper psychiatric follow-up is crucial to improve future outcomes.

Hence, patient care in the case of pretilachlor poisoning has a very different approach compared to the more commonly encountered OP compound poisoning. Adequate history and symptom identification, along with supportive laboratory values, are key to appropriate diagnoses. Treating physicians must always keep pretilachlor as a probable agent in mind when dealing with unknown herbicide/pesticide poisoning

Conclusion

This case report highlights the importance of early identification of pretilachlor poisoning. An awareness of this commonly available herbicide and understanding its clinical presentation, which closely mimics OP poisoning, is essential. It also stresses the avoidance of aggressive treatment with intravenous atropine/pralidoxime in an unconfirmed chemical compound poisoning case.

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