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A Case Series on 2, 4-Dichlorophenoxyacetic Acid Poisoning Mimicking Organophosphorus Toxicity: Treatment Approaches and Outcomes

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Abstract: ***Background**: 2, 4-Dichlorophenoxyacetic acid (2, 4-D) is a widely used herbicide in agricultural regions, particularly in South Asia. Its ingestion, often with suicidal intent, can closely mimic organophosphorus (OP) poisoning, resulting in frequent misdiagnosis and delayed targeted therapy. Limited clinical literature exists on the diagnostic and therapeutic approach to 2, 4-D toxicity. **Objective**: To describe the clinical presentation, diagnostic challenges, treatment strategies, and outcomes in a series of patients with 2, 4-D poisoning, with a focus on differentiating it from OP toxicity and emphasizing the role of urinary alkalinization. **Methods**: We retrospectively studied cases of 10 patients with confirmed or strongly suspected 2, 4-D poisoning admitted to the emergency department of Government Medical College, Miraj, Maharashtra (India). Clinical data including presenting symptoms, laboratory findings, treatment interventions, and outcomes were analyzed. **Results**: All patients presented with gastrointestinal and neuromuscular symptoms mimicking of OP poisoning. However, preserved Glasgow Coma Scores, normal plasma cholinesterase levels, and lack of response to atropine/pralidoxime raised suspicion for alternative diagnoses. Metabolic derangements, including hypernatremia and elevated anion gap metabolic acidosis, were prominent. Early initiation of urinary alkalinization and supportive therapy led to full recovery in all cases, with no ICU admissions or fatalities. **Conclusion**: 2, 4-D poisoning is a frequently misdiagnosed yet clinically distinct toxidrome that requires high diagnostic vigilance. Metabolic abnormalities and neuromuscular signs provide critical diagnostic clues. Early urinary alkalinization is a safe, effective adjunct that may significantly improve outcomes. Emergency clinicians should maintain a high index of suspicion to avoid therapeutic misadventures in cases of unknown agricultural poisoning.*

Keywords: 2, 4-D poisoning; organophosphorus mimicry; metabolic acidosis; urinary alkalinization; herbicide toxicity; emergency medicine; misdiagnosis

1. Introduction

The chlorophenoxy herbicide, 2, 4-dichlorophenoxyacetic acid (2, 4-D) is widely used in agriculture, particularly in regions with extensive cereal crop cultivation such as Western India. Despite its widespread use, cases of human poisoning remain relatively underreported. When ingestion occurs, often with suicidal intent due to its easy availability, it presents a unique diagnostic and therapeutic challenge in emergency medicine[1].

One of the major clinical concerns surrounding 2, 4-D poisoning is its presentation mimicking organophosphorus (OP) compound toxicity, a much more common form of agrochemical poisoning in South Asia. Patients may present with miosis, salivation, vomiting, altered sensorium, and muscle fasciculations—features that closely resemble the cholinergic toxidrome as seen in OP poisoning[2]. As a result, many cases are misdiagnosed and treated empirically with atropine and oximes, which are ineffective in 2, 4-D poisoning and may lead to delays in appropriate treatment[3].

Unlike OP compounds that inhibit acetylcholinesterase, 2, 4-D toxicity primarily affects skeletal muscle and the central nervous system, often resulting in rhabdomyolysis, coma, myotonia, and metabolic acidosis. The herbicide is thought to cause mitochondrial dysfunction and disrupt cellular respiration, contributing to its multisystem toxicity[4]. These distinct pathophysiological mechanisms underscore the importance of early and accurate diagnosis.

There is currently no specific antidote for 2, 4-D poisoning. Management is primarily supportive, with evidence suggesting a beneficial role for urinary alkalinization, forced diuresis, and in severe cases, haemodialysis[5, 6]. Given the herbicide's low protein binding and renal excretion, enhancing its elimination can significantly alter outcomes in acute toxicity.

This case series aims to contribute to the limited clinical literature on 2, 4-D poisoning by presenting a series of patients initially misdiagnosed as OP poisoning but ultimately managed effectively with specific supportive strategies. We emphasize the critical importance of diagnostic differentiation, detailed exposure history, and supportive treatment protocols in improving patient outcomes in this frequently misrecognized but potentially lethal toxidrome.

2. Methods

Study Design: This case series was conducted as a retrospective observational study evaluating the clinical

presentation, management strategies, and outcomes in patients with confirmed 2, 4-dichlorophenoxyacetic acid (2, 4-D) poisoning over a period of 6 months.

Study Setting: The study was carried out in the Emergency Medicine Department and Intensive Care Unit of GMC Miraj, a tertiary care teaching hospital in Maharashtra, India, which serves as a regional referral centre for toxicology and acute medical emergencies.

Inclusion Criteria: Patients were included if they fulfilled the following criteria:

- Documented history or witnessed ingestion of a 2, 4-D-containing compound
- Confirmation of 2, 4-D exposure via either retrieval of the container, clinical corroboration of symptoms, or toxicological screening
- Complete clinical records available for review

Exclusion criteria:

- Patients of age less than 12 years.
- Poisoning with other compounds along with 2, 4-D

Ethics statement: The study was reviewed and approved by the institutional Ethics Committee of GMC Miraj, Maharashtra. All identifying patient data were anonymised.

3. Results

In our study a total of 10 case were enrolled out of which 6 were male and 4 were female, aged 19 to 87 years, who presented within 1–2 hours after ingesting 10–70 mL of 2, 4-Dichlorophenoxyacetic acid (2, 4-D). Common symptoms included vomiting, abdominal pain, loose stools, myalgia, generalized weakness, and in one case, muscle fasciculations was present. All patients had stable vital signs on admission, with GCS ranging from 12 to 15. Laboratory investigations, including renal and liver function tests, were within normal limit. Arterial blood gas (ABG) analyses three cases showed mild to moderate metabolic acidosis, with anion gaps ranging from 10.8 to 28. Plasma cholinesterase levels, measured in most patients, were within normal limits (5000–7500 U/L).

Management was primarily supportive, involving gastric lavage, activated charcoal, intravenous fluids (normal saline or lactated Ringer's), and symptomatic treatment. Three patients received alkaline diuresis, and none required mechanical ventilation or dialysis, except for one case with reduced urine output. All patients recovered without complications and were discharged within 3–5 days. This series highlights that 2, 4-D poisoning, even at moderate doses, typically results in a favorable outcome with early decontamination and supportive care.

Table 1: Summary of Clinical Characteristics and Outcomes in 10 Cases of 2, 4-D Poisoning

Case	Age	Gender	Time to Hospital (hrs)	Dose Ingested (ml/mg)	pH	Anion Gap	Cholinesterase (U/L)	Hospital Stay (days)	Outcome
1	24	Female	2.0	10–20	7.38	12.5	6246	4	Discharged
2	25	Female	1.0	20	7.35	9.0	6231	3	Discharged
3	19	Female	1.0	30	7.38	28.0	6500	3	Discharged
4	30	Female	1.5	15	7.30	10.8	6520	3	Discharged
5	25	Male	1.0	20	7.35	20.0	7500	3	Discharged
6	45	Male	2.0	45	7.23	27.3	5000	5	Discharged
7	55	Male	1.0	20	7.32	21.0	6500	3	Discharged
8	35	Male	1.0	70	7.28	23.0	6500	3	Discharged
9	87	Male	1.5	30	7.28	21.0	7580	4	Discharged
10	35	Male	2.0	30	7.32	25	7000	4	Discharged

4. Discussion

In acute emergency settings, 2, 4-Dichlorophenoxyacetic acid (2, 4-D) poisoning often presents with a toxidrome indistinguishable from organophosphorus (OP) poisoning, leading to frequent misdiagnosis and inappropriate early treatment. Symptoms such as vomiting, miosis, muscle fasciculations, bradycardia, and altered consciousness mimic cholinergic toxicity resulting in reflex administration of atropine and pralidoxime[1] however 2, 4-D causes direct muscle toxicity and uncouples oxidative phosphorylation. This "therapeutic anchoring" has been reported even in tertiary care settings, as illustrated by multiple cases of misdiagnosed 2, 4-D poisoning initially treated as OP ingestion [3].

Key Differentiators from Organophosphorus Poisoning: Differentiating 2, 4-D toxicity from OP compounds is clinically essential, especially when exposure history is unreliable. While OP toxicity is cholinergic in mechanism (AChE inhibition), 2, 4-D affects the central and peripheral nervous system through mitochondrial dysfunction and direct myotoxicity, leading to rhabdomyolysis, hypertonia or hypotonia, and even paralysis[4, 5]. Key distinguishing features include:

- Preserved or mildly depressed GCS despite high doses[7, 8] unlike OP poisoning in which it is mainly due to central cholinergic overstimulation.
- Normal plasma cholinesterase levels, unlike depressed levels in OP poisoning[9].
- Ineffectiveness of atropine/pralidoxime, reinforcing a non-cholinergic mechanism[2] less than 4 hours.
- Neuromuscular rigidity or myotonia, which are not typical features of OP poisoning[1, 10, 11]. Recognizing these features early is crucial to avoid therapeutic delays and misdirection.

Metabolic Abnormalities: A Diagnostic and Prognostic Window: One of the striking features in our series was the presence of metabolic acidosis with elevated anion gap (up to 28) in more than half the patients. This is consistent with published cases, where 2, 4-D was found to cause metabolic acidosis, renal tubular damage, and disruption of mitochondrial oxidative phosphorylation[12, 13]. Hypernatremia, observed in multiple cases in our series, may result from both dehydration due to vomiting and possible

renal sodium handling dysfunction. However, it is not typical in organophosphate (OP) poisoning and, if present, usually occurs secondary to fluid loss. These metabolic patterns can serve as important diagnostic clues when the identity of the poison is uncertain.

Therapeutic Strategy: Emphasis on Urinary Alkalinization: There is no specific antidote for 2, 4-D toxicity. However, urinary alkalinization and forced alkaline diuresis have shown consistent clinical benefit and are widely endorsed in toxicology literature.2, 4-D, being a weak acid with low protein binding and high renal clearance, Making the urine alkaline helps the body get rid of the poison faster.6, [14]. In our cases, patients receiving early urinary alkalinization had rapid recovery without need for urgent haemodialysis, even in higher-dose exposures (e. g., Case 8, 70 mL ingestion). Prompt urinary alkalinization is a key therapeutic strategy in 2, 4-D poisoning, unlike organophosphate (OP) poisoning, which requires atropine and pralidoxime. This underscores the importance of maintaining a high index of suspicion in patients with preserved Glasgow Coma Scale (GCS) scores and highlights the need for immediate estimation of cholinesterase levels at admission.

Despite the potential lethality of 2, 4-D compounds, all patients in our cases survived, with short hospital stays and no ICU admissions. This outcome is especially noteworthy in contrast to literature reports of severe toxicity, mechanical ventilation, and even death, often linked to diagnostic delay or lack of directed treatment[2, 12]. Our findings reinforce the importance of early identification, careful clinical differentiation from OP poisoning, and prompt initiation of urinary alkalinization and supportive care.

5. Limitations of the Study

1) Definitive toxicological confirmation of 2, 4-D exposure via serum or urine assays was unavailable in all cases.
2) Being retrospective study, no post-discharge follow-up or creatine kinase testing data was available, also limited data about assessment of delayed effects and muscles injury.

6. Conclusion

2, 4-Dichlorophenoxyacetic acid (2, 4-D) poisoning can present with gastrointestinal and neurological symptoms. In our cases, patients showed mild to moderate metabolic disturbances—acidosis and hypernatremia—suggesting systemic toxicity due to kidney and mitochondrial involvement. All patients recovered with supportive care and adjunctive treatment such as urinary alkalinization. Early diagnosis, public awareness, and proper labeling are key to reducing the risk. Further dedicated studies are needed to corroborate the findings of this case series.

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