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Case Study

DICHLORODIPHENYLTRICHLOROETHANE (DDT) POISONING: A CASE REPORT

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ABSTRACT

Objective: Dichlorodiphenyltrichloroethane (DDT) is a highly lipophilic organochlorine pesticide known for its environmental persistence and neurotoxicity. Although banned or restricted in many countries, sporadic cases of poisoning continue to occur due to illegal use and accidental exposure. Acute toxicity primarily affects the nervous system.

Methods: We report a case of acute DDT poisoning in a 34 y old male who presented with gastrointestinal and neurological symptoms following accidental ingestion. The patient initially had stable vital parameters but subsequently developed respiratory failure and hypotension requiring mechanical ventilation and vasopressor support. Management was entirely supportive, as no specific antidote exists for DDT toxicity.

Results: The patient showed progressive clinical improvement and was successfully extubated on the third day, with complete recovery.

Conclusion: DDT poisoning, though uncommon, should be considered in patients presenting with unexplained neurological manifestations and a relevant exposure history. Early recognition and aggressive supportive management are crucial for favorable outcomes.

Keywords: DDT poisoning, Organochlorine pesticide, Neurotoxicity, Respiratory failure, Case report

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INTRODUCTION

Dichlorodiphenyltrichloroethane (DDT) is a synthetic organochlorine insecticide previously used extensively for agricultural and vector control purposes. Due to its lipophilic nature, it accumulates in adipose tissue and biomagnifies through the food chain, leading to long-term environmental and human exposure [1].

DDT exerts its toxic effects primarily by altering voltage-gated sodium channel kinetics, resulting in prolonged depolarization and neuronal hyper excitability [2, 3]. Clinical manifestations range from mild gastrointestinal disturbances to severe neurological complications including tremors, seizures, and coma.

Although its agricultural use has been banned in many countries, cases of poisoning continue to be reported in developing regions due to illegal usage and improper storage practices [4, 5]. We report a case of acute DDT poisoning presenting with delayed respiratory compromise requiring ventilatory support.

Case presentation

A 34 y old male with no known comorbidities presented to the emergency department with nausea, vomiting, abdominal pain, dizziness, and generalized weakness for six hours. These symptoms were followed by tremors and restlessness.

On detailed history, the patient reported accidental ingestion of a pesticide stored in an unlabelled household container. The substance was later identified as DDT powder used illegally for pest control. There was no history of alcohol intake, substance abuse, or suicidal intent.

On arrival, the patient was conscious, oriented, and obeying commands. Vital parameters were:

Pulse – 108/min

Blood pressure – 130/80 mmHg

Respiratory rate – 22/min

SpO_2 – 98% on room air

Gastric lavage was performed with normal saline, and the patient was managed conservatively under close observation.

Within 24 h, the patient developed drowsiness, hypotension (BP 84/56 mmHg), tachypnea (RR 38/min), and hypoxia (SpO_2 70%). Arterial blood gas analysis revealed:

pH – 7.29

pCO_2 – 65 mmHg

pO_2 – 80 mmHg

HCO_3^- – 29 mEq/l

Lactate – 0.8 mmol/l

He was immediately intubated and mechanically ventilated. Intravenous fluids were administered, and noradrenaline infusion was initiated and titrated to maintain adequate mean arterial pressure.

There is no specific antidote for DDT poisoning. Unlike organophosphorus poisoning, atropine and pralidoxime have no therapeutic role [2, 6, 7].

The patient showed gradual clinical improvement. He was successfully extubated on the third day and shifted to high-flow oxygen therapy. Continuous monitoring for three additional days revealed stable hemodynamics and normal arterial blood gas parameters. He was subsequently shifted to the ward and discharged in stable condition.

DISCUSSION

DDT toxicity primarily affects the central nervous system by prolonging sodium channel opening, resulting in repetitive neuronal firing and hyperexcitability [3, 8]. Clinical features include gastrointestinal symptoms, tremors, seizures, altered sensorium, and, in severe cases, respiratory depression and cardiovascular instability [1, 2].

Diagnosis is largely clinical and based on exposure history, as laboratory confirmation is often unavailable, particularly in resource-limited settings. Management remains supportive, focusing on airway protection, seizure control, hemodynamic stabilization, and monitoring for delayed neurological deterioration.

Unlike organophosphorus poisoning, cholinergic manifestations are absent, and specific antidotes such as atropine or pralidoxime are ineffective [6]. Prognosis is generally favorable with early recognition and prompt supportive care.

Continued illegal availability and unsafe storage of banned pesticides remain significant public health concerns in developing countries [4, 9]. Increased regulatory enforcement and public education are necessary to prevent such incidents.

CONCLUSION

Although rare, DDT poisoning should be considered in patients presenting with acute neurological symptoms and a history of pesticide exposure. Timely supportive management, including airway protection and hemodynamic stabilization, can lead to complete recovery. Strengthening public awareness and pesticide regulation is essential to reduce preventable toxic exposures.

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AUTHORS CONTRIBUTIONS

All authors have contributed equally

CONFLICTS OF INTERESTS

The authors declare no conflicts of interest

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