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A case report on 2,4 dichlorophenooxyacetic acid poisoning

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Abstract

2,4-Dichlorophenoxyacetic acid (2,4-D) is a herbicide used by agriculturists. It has a very high toxic effect. Management is only supportive with alkaline diuresis and treatment of complications as there is no specific antidote to this compound. Complications occur due to difficulty in the diagnosis or delay in the diagnosis.

Keywords: Herbicides, alkaline diuresis, fatal

Introduction

2,4-D is a herbicide of chlorophenoxy herbicide group widely used in northern India against weeds in cereal crops, lawns and parks. Ingestion, skin contact and inhalation are three main route of human exposure to 2,4-D herbicides.[1] There are very few documented cases with this compound poisoning have been reported. Chances of wrong or misdiagnosis are high as initial signs and symptoms can mimic that of anticholinesterase poisoning. There is no specific antidote for 2,4-D herbicide poisoning.[2]

Acute lethal levels of 2,4-D in the plasma appear to lie between 447 mg/liter and 826 mg/liter. Its toxic effects involve all the organs and systems of body,[3] and Blood levels of 2,4-D can be measured most accurately with gas-liquid chromatography with electron-capture detection.

Case description

A 36-year-female patient presented with complaints of nausea, vomiting without blood, abdominal pain, blurring of vision and loss of consciousness after 2 hours of oral ingestion of about 250 mL of a herbicide with brand name of Cloud 58 whose active ingredient is 2,4-D ethyl ester. However, there was no fasciculation, Diarrhoea, lacrimation/salivation or lung crepitation's.

Her blood pressure was 100/60 mm Hg and pulse rate was 118/min. Respiratory rate was 20/min and pupils were mid dilated and reactive. Bilateral chest auscultation revealed vesicular breath sounds. ECG showed sinus tachycardia. Initial neurological assessment revealed Glasgow coma scale (GCS) of 10. Immediately gastric lavage with charcoal was done. She complained of blindness. Vision was found to be perception of light only.

Her hemoglobin was 10.4 mg/dL, total leukocyte count was 19000, and platelet count was 1.88 lakh/mm^3. Her liver and renal functions were normal. ABG showed metabolic alkalosis. Blood levels of 2,4-D could not be measured as the required facility was not available.

This poison does not have any specific antidote. Literature search resulted in finding a role of forced alkaline diuresis in its management. We started treatment with intravenous fluids, sodium bicarbonate and injectable furosemide to cause forced diuresis.

Gradually sensorium improved and urine output was maintained. Tachycardia resolved after 4 hours. Next day, patient was hemodynamically stable with a GCS of 15. But vision did not improve.

Alkaline diuresis and other treatments were continued. Ophthalmology consultation was done and post discharge ophthalmology treatment was planned. The patient was discharged from hospital after 3 days of admission being hemodynamically stable and with normal laboratory tests.

Discussion

The principle routes of exposure of phenoxy compounds are pores and skin, inhalation and oral ingestion. The exact mechanism of phenoxy herbicides toxicity is doubtful. These compounds are irritants and reason corrosive results in gastrointestinal tract inflicting nausea, vomiting, abdominal or throat pain and diarrhea. They also motive neuromuscular toxicity and myotonia via inhibition of the voltage-gated chloride channel (CLC-l) in skeletal muscular tissues.[4]

Metabolic acidosis, hyperventilation, hyperkalemia, hyperthermia, elevated creatine kinase, generalized muscle rigidity, hypotension or asystole are criteria of excessive toxicity.[5]

Various mechanisms of toxicity such as dose-based mobile membrane harm, uncoupling of oxidative phosphorylation and disruption of acetyl coenzyme A metabolism had been proposed.

Unconsciousness or coma may end result from a right away CNS depressant action or metabolic derangements in patients. Hypertonia, hyperreflexia, ataxia, nystagmus, miosis, hallucinations, convulsions, fasciculation and paralysis might also gift at variable periods throughout the direction of systemic toxicity.[6]

Our case confirmed suitable response with urine alkalinization. Urine alkalinization is one form of enhance removal that may be useful in a few poisoning inclusive of phenobarbital, chlorpropamide, salicylate, chlorophenoxy herbicides especially 2,four-dichlorophenoxy acetic acid and mecoprop.[5] on account that myoglobinuria produces nephrotoxicity, alkaline diuresis additionally can be helpful in stopping renal damage[7]. Chlorophenoxy herbicides are vulnerable acids (pKa = 2.6 for two,four-D) and excreted inside the urine unchanged. Alkaline diuresis particularly in severe 2,4-D poisoning can be lifesaving.[8]

Plasma alkanalization might also limit the distribution of phenoxy compounds from the important flow by way of ion trapping.[7,9]

The poison is exceedingly fatal with the mortality achieving almost a hundred% in the reports from the rest of the sector.[10] our patient thankfully had only gastrointestinal results at presentation and no pulmonary, renal or myotoxic consequences. Possibly, vomiting after ingestion helped the patient in lowering absorbed dose of poison and pressured alkaline diuresis initiated helped in rapid recovery of our patient.

Conclusion

It is a probably deadly poison and the handiest manner of identifying the poison is to have a look at the field or the literature to be had with the field. Clinical presentation can mimic of these of anticholinesterase poisoning so a careful look for presence of signs and symptoms of corrosive harm, muscle weak spot (proximal > distal) and neuropathies will assist in identifying instances with 2,4-D ingestion. Excessive toxicity symptoms ought to be in

thoughts and an alkaline diuresis can increase herbicide elimination.

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